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STUDIES OF THE ACTIVITY OF THE GRAVID HUMAN UTERUS*

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THE STUDIES on the behaviour of the gravid human uterus were begun in 1950 in an attempt to further the knowledge of uterine function in labour. At the outset of this study a tokodynamometer (TKD) was obtained with the intention of studying primary uterine inertia but during the initial phases of the work it was realized the problem would need a broader scope in face of the large background of material available from such investigators as Blair-Bell,[3] Moir,[19] Nixon,[23] Murphy,[21] Reynolds,[29] and many others.

This research falls into three distinct parts. The first studies were done on strips of uterine muscle removed from gravid uteri at Cæsarean section, and are concerned with effects of various ions on the uterus *in vitro*. In conjunction with this, blood from parturient women was investigated for changes in these ions. The second part of the study was done with the aid of the TKD. In this phase we tried to correlate some of the observations recorded by the TKD and the clinical picture of the patient in labour, with a view to using the TKD as a means of differentiating various types of labours. The last section of the work was prompted by a very timely paper written in 1952 by Halliday and Heyns[12] of South Africa concerning the lack of correlation between electrical TKD methods of recording contractions. In the third part of the paper an attempt has been made to show what is being measured by the TKD by using a model of a gravid uterus.

I. IN VITRO STUDIES ON HUMAN GRAVID UTERINE MUSCLE

Method.—Small strips of uterus were removed from both upper and lower segments of the uterus at five different Cæsarean sections in cases where trial labour had been in effect. These were transferred immediately to cold Locke's solution and within one-half hour put in an oxygenated constant temperature bath. Records of rhythmic movements were taken for about one hour and then the ion concentrations of the bath were varied.

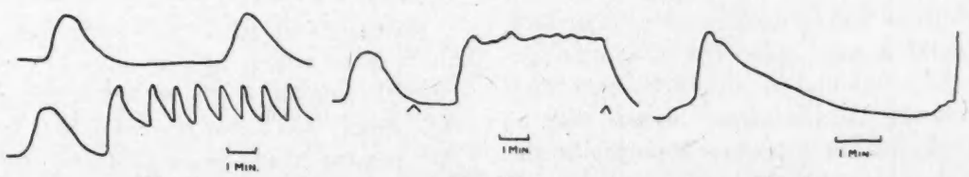

Fig. 1 Fig. 2 Fig. 3

Fig. 1.—The top record shows the contractions of a strip of gravid uterus in Locke's solution. The lower record shows the effect of an excess of potassium ion in Locke's solution. Fig. 2.—Showing the effect of a potassium-deficient Locke's solution on a strip of gravid uterus. Fig. 3.—Effect of an excess of calcium in the Locke's solution on a strip of gravid uterus.

Results.—Potassium was gradually increased from the concentration of 5.64 m. eq./l. in Locke's solution to 14.3 m. eq./l. by adding KCl to the bath and removing chloride ions from the sodium content to keep the total ion concentration the same. The excess potassium caused a definite increase in both tone of the muscle and frequency of contractions, giving a clonic pattern. The pattern varied with the concentration to a maximum clonus with three times the concentration found in the blood (Fig. 1). When a potassium-deficient solution was used, a similar effect was produced on the strip as is seen in Fig. 2.

The effect of an excess of the calcium ion was to increase the amplitude and decrease the frequency of the contractions, while the tone remained the same. Calcium concentration was increased from the 2.19 m. eq./l. in Locke's solution to 4.63 m. eq./l. (Fig. 3).

A lack of calcium relaxed the muscle and an excess of magnesium had a slight relaxant effect. Magnesium was added in the form of magnesium chloride to the maximum of 16.2 m. eq./l. to produce this effect.

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DISCUSSION

Very little work has been done on the effects of various ions on the gravid human uterus, but the work that has been done has produced varying hypotheses. The ion content of the uterus is different from other muscles of the body since it contains more sodium and calcium but less potassium, magnesium and phosphorus. The functional effect of this difference has never been fully explained.[29]

Speculations about the rôle of calcium and potassium on uterine motility have been advanced and these hypotheses are based on the knowledge that these ions usually affect protoplasmic activity. In general, calcium has been considered a stimulant and potassium and magnesium have been considered depressants. While these observations may be true in cardiac muscle, in the study presented here, calcium had a depressant action, inasmuch as it decreased the frequency of the contractions although it increased the amplitude of the contractions. Potassium had an excitatory effect in excess or deficient amounts and only magnesium acted as a depressant as it does on other types of muscle. Rossenbeek[30] has shown the changes that occur in the calcium-magnesium ratio in rabbits during gestation. He found that near term the ratio approaches a level that favours motility. Blair-Bell, Datnow and Jeffcoate[3] place the same interpretation on the potassium-calcium ratio in women during gestation. Others have shown that as labour progresses the potassium increased in the blood and the calcium decreases.[31]

As a supplement to the *in vitro* study, blood was taken from a number of patients in various stages of labour at the same time as TKD tracings were made, to see if there was any change in the ionic content of the blood to correlate with the *in vitro* experiments. However, no changes were noted in the concentration of potassium, magnesium or calcium using a colorimetric method of analysis. The blood findings in this study agree with those of Parvianinen[24] but differ with the results of others as cited above. Part of the explanation of this discrepancy may be found in the work of Bodansky[4] and his associates who have shown that the calcium content of the blood in pregnancy varies with the season of the year and that really two normal levels should be used. Newman[22] has shown there is a slight fall in calcium concentration during pregnancy to the ninth month when it begins to rise until it is back to normal levels in the puerperium. Furthermore, it should be kept in mind that the colorimetric measurement of serum potassium is difficult and the changes in the serum are slow and may not be an absolute indication of the ion level in the cells.

Similarly no change was found in the ion concentration of the three elements under investigation in blood samples taken during abnormal types of labour. In all cases the values were within normal limits.

Another source of error was in the selection of the site for withdrawing the blood. The blood was taken from an arm vein and since the venous blood reflects the metabolism of the part it is draining, the blood should have been taken from the uterine vein in order to assess the changes that might be brought about by the action of the uterus in labour.

One group of workers[25] approached the problem by injecting calcium into patients, and they found that they got a varying response with the same dose in different patients. If there was a response, it shortened the time between contractions but had no effect on the length of contraction. No assessment was made of the height or strength of contraction. This observation is at variance with the *in vitro* experiments.

Hastings and van Dyke[13] state that calcium is the ion to which uterine muscle is most susceptible but the work on which this conclusion was based was done on animals. The result of the present study would suggest that uterine muscle is more susceptible to changes in the potassium concentration. Injection techniques have also shown that magnesium will relax a tetanically contracting uterus.[1] In this work magnesium sulphate was injected into women who were suffering tetanic contractions produced by oxytocics. Magnesium has long been one of the standard drugs used in toxæmia of pregnancy to reduce the cellular irritability.

CONCLUSIONS

From the above experiments and observations on strips of gravid human uterus and on blood from women in labour, it would seem that the following conclusions can be drawn from the *in vitro* experiments:

1. An excess of potassium ion produced the same effect as a deficiency of the ion, namely, an increase in tone and frequency of contractions.

2. An excess of calcium increased the amplitude and decreased the tone and frequency of contractions.

3. Excess magnesium had only a slight effect on reducing the tone when given in high concentrations.

4. The greatest response was seen in relation to changes in potassium ion concentration, contrary to observations on other types of mammalian muscle.

5. No change was found in the concentration of potassium, calcium or magnesium ions in the serum of women during labour taken from arm vein blood.

II. TKD RECORDS—CORRELATED WITH TYPES OF LABOUR

Before the advent of the multi-channel tokodynamometer of Reynolds, Murphy[21] made many observations with the Lorand Tocograph which records from only one point on the abdomen at a time. He laid emphasis on the ability to distinguish types of labour by the smoothness, height of the wave, frequency and regularity of the contractions.

Most of the recent work dealing with the recording of uterine contractions and analysis of these records has come from Reynolds and his co-workers at Johns Hopkins and the Carnegie Institution of Washington. Reynolds[27] states that the criteria of efficient uterine contractions are: (1) fundal dominance; (2) sustained contractions; and (3) contractions of sufficient magnitude. Later the criteria were increased to include a decreasing gradient of activity toward the lower segment.[14] Alvarez and his group supplemented these concepts by recording pressure changes of the amniotic fluid by means of a manometer connected to a needle which was pushed through the anterior abdominal wall.[2, 7] They found that the measurement of internal pressures and the pattern of the external recording by the multichannel tokodynamometer and the polygraph in normal and abnormal labours could be correlated.[7]

Neither of the above authors has emphasized the rate of rise of uterine contractions in either normal or abnormal labours although Reynolds[26] states he records it.

MATERIAL AND METHODS

The patients used in this investigation were chosen at random from primigravidæ admitted to the wards of the Kingston General Hospital. Tracings were taken by means of a three-channel tokodynamometer (TKD), as shown in Fig. 4, which is similar to the one used by Reynolds and co-workers.[26] Fifty-two records were made on 49 patients. Each record was of at least 20 minutes' duration and the transducers were placed in areas B, E and H, that is, in the midline above the umbilicus, just below the umbilicus and above the pubis.[26] The tracings were all

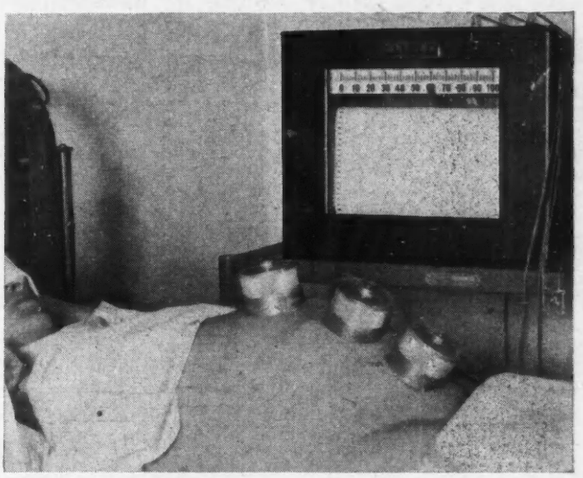

Fig. 4.—The Tokodynamometer at the time of a tracing with the strain gauges in position on the patient.

made during the first stage of labour and the patients had received either Demerol or morphine as analgesics but none of them had had any type of anæsthesia.

The tracings were analyzed for frequency, duration, dominance and rate of rise of contractions. While the rate of rise of contractions, or rate of contraction, in the fundus is due to displacement caused by the uterine contractions, the relaxation phase or rate is a complex curve made up of the readjustment of abnormal viscera upon cessation of the uterine contractions. This produces a variable curve that frequently has a fast and slow component, hence the pattern is very irregular. This was also found to be the case in the relaxation curve by Reynolds,[26] so for these reasons we did not record the rate of relaxation in the fundus.

The contraction rate (by which is meant rate of rise of the fundal contraction) was calculated by dividing the average height, in inches, of the fundal contraction by the average time, in seconds, from the point where the record of the contractions first started to its peak. The TKD was always calibrated so that depression of the standardizing button produced a scale deflec-

tion of 12.5 units which is equivalent to an applied force of 2 ounces (56.70 gm.) on the transducer.

Each case was re-assessed after delivery and the clinical course of the labour discussed and classified. The cases were then grouped into: (1) false labour, which either stopped for a period of time or continued into true labour (prodromal labour); (2) primary uterine inertia, and (3) normal labour. The clinical criteria used for false labour and primary uterine inertia are those described in standard texts such as those of DeLee and Greenhill,[8] Eastman,[11] Titus,[33] McCormick,[18] etc.

be false labour, primary uterine inertia, or normal labours on clinical appraisal.

Table I shows the record number (which corresponds to the number under the point in Fig. 5), position, rhythm, rate, dominant part of the uterus, presence or absence of progressive dilatation and effacement of the cervix, clinical diagnosis and total hours of labour.

DISCUSSION

Tracings have been taken with the TKD in this hospital for three years and similar patterns have been observed as have been reported by various investigators. It was felt that analysis of

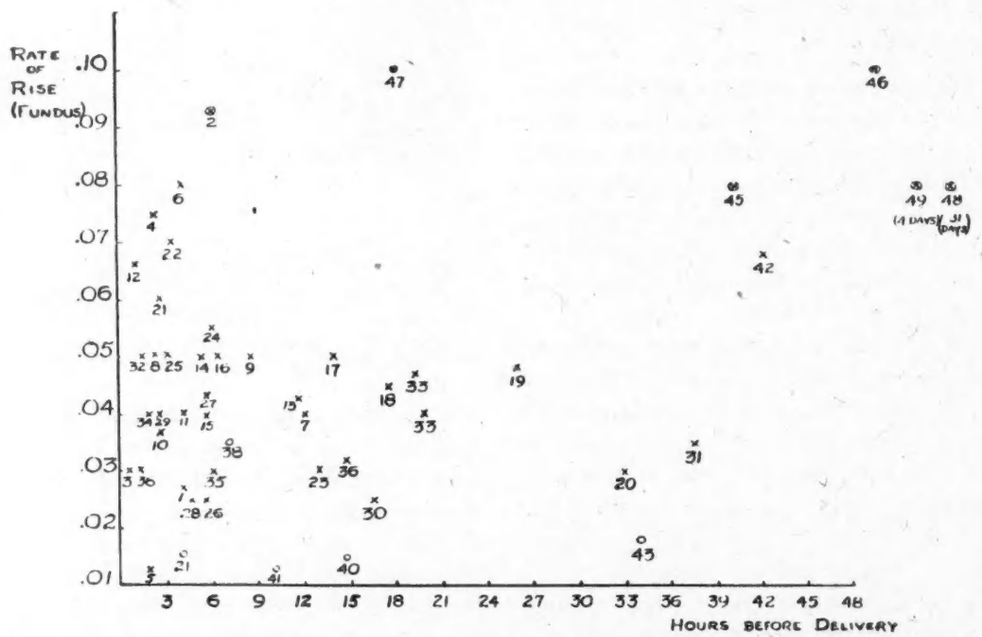

Fig. 5.—A graph showing the rate of rise of the fundus, ordinate, plotted against the time before delivery, abscissa. The numbers under the points refer to the record numbers of Table I. The clinical diagnosis is shown as follows: (X) False Labour, O Primary Uterine Inertia and X Normal Labour.

This part presents the correlation between the clinical classification of the labour and the analysis of the tokograms (TKG's).

RESULTS

Many patients have periods of false labour or prodromal labour before true labour begins, so it is often difficult to determine the definite time of onset of true labour. Hence the time of birth was used as a reference point in the various calculations.

The graph in Fig. 5 shows the relationship of the rate of contraction in the fundus to the hours before delivery. The symbols above the points on the graph indicate cases that were considered to

these records in the usual way for frequency, regularity, co-ordination and dominant segment often did not enable the prediction of the type of labour that could be expected. The records were then re-analyzed and it was postulated that the uterine muscle would have an optimal rate of contraction when the greatest work would be done in dilating the cervix and expelling the fetus. The rates of contraction of the TKG's were calculated and it was found that there was a range of optimal rate of contraction in the fundus. This rate alone was not always significant but used in conjunction with the other factors usually measured from the tracings, it was then possible to distinguish three types of labour

patterns: primary uterine inertia, false labour and normal or average labour.

Fig. 5 shows the rates of uterine contraction in the fundus plotted against the hours before delivery. From this graph it is seen that the rates fall into three categories: those greater than 0.08 inches per second, which clinically were assessed as false labour; rates of under 0.018 inches per second which were diagnosed as primary uterine inertia; and those tracings with rates between these two limits which were labour of average duration and characteristics.

Table I shows the rates of contraction and the clinical diagnosis in conjunction with the other factors usually taken from TKG's.

Records 7 and 49 are from the same patient. Record 49 was taken 4 days before labour began. The patient later came into hospital in true labour and delivered herself within 18 hours. Record 7 was made 12 hours after active labour started. In this case it will be seen that the true labour contractions are regular and at a lower rate as compared to the false labour contractions exhibited 4 days before. Fig. 6 shows typical contractions of this patient in both false and true labour. In the other cases of false labour, records 45 to 49 inclusive, the rates are high. In two records, namely 47 and 49, the rhythm is irregular and the patterns are similar to those described by Murphy[20] as "pregnancy type" of false labour. Only in one case, record 48, was there a lack of fundal dominance as described by Reynolds.[27] In this case the fundal contractions equalled the mid-zone contractions but the rate was greater than normal labour.

Records 38 to 44 are of cases classified as primary uterine inertia. All of these cases have slow rates of contraction in the fundus except record 38. This record showed all the criteria necessary for an efficient labour. The contractions of the fundus were dominant, sustained and co-ordinated with the mid-zone. The lower segment was inactive. However, the fetus was presenting by the breech. This case would fit into the type of inertia caused, in part at least, by poor apposition of the breech to the cervix.[27] This patient was finally delivered of a living male infant weighing 9 pounds, 6 ounces (4,252.5 g.) by Cæsarean section.

TABLE I.

SUMMARY OF DATA FROM TKG'S.

Record number	Pos'n.	Rhythm	Rate (inches per second) fundus	Dominant zone	Dilat. and effacement	Total hours labour	Clinical assessment
1	LOA	reg.	0.028	F	yes	13:08	labour
2	LOA	reg.	0.093	F	?	10:30	labour
3	LOA	reg.	0.030	F	yes	10:30	labour
4	LOA	irreg.	0.075	F	yes	10:20	labour
5	LOA	reg.	0.012	M	yes	5:35	labour
6	LOA	reg.	0.080	F	yes	9:15	labour
7	LOA	reg.	0.040	F	yes	18:10	labour
8	LOA	reg.	0.050	F	yes	7:55	labour
9	LOA	reg.	0.050	F	yes	10:01	labour
10	LOA	reg.	0.037	F	yes	17:06	labour
11	LOA	reg.	0.040	F	yes	8:18	labour
12	LOA	reg.	0.066	F	yes	11:00	labour
13	LOA	reg.	0.043	F	yes	20:00	labour
14	LOA	reg.	0.050	F	yes	11:40	labour
15	LOA	reg.	0.040	F	yes	19:47	labour
16	LOA	reg.	0.050	F	yes	29:37	labour
17	LOA	reg.	0.050	F	yes	25:35	labour
18	LOA	reg.	0.045	F	yes	40:20	labour
19	ROA	reg.	0.048	F	yes	36:28	labour
20	ROA	reg.	0.060	F	yes	13:13	labour
21	ROA	reg.	0.070	F	yes	9:26	labour
22	ROA	irreg.	0.040	F	yes	44:00	disproportion
23	ROA	reg.	0.030	F	yes	44:00	labour
24	ROA	reg.	0.055	F	yes	25:00	labour
25	ROA	reg.	0.055	F	yes	9:00	labour
26	ROA	reg.	0.025	F	yes	23:25	labour

TABLE 1.—*Continued*

SUMMARY OF DATA FROM TKG'S.

Record number	Pos'n.	Rhythm	Rate (inches per second) fundus	Dominant zone	Dilat. and effacement	Total hours labour	Clinical assessment
27	ROA	reg.	0.043	F	yes	13:30	labour
28	ROA	irreg.	0.025	F	yes	26:30	labour
29	LOP	reg.	0.040	F	yes	6:05	labour
30	LOP	reg.	0.025	F	yes	35:30	labour
31	LOP	reg.	0.035	F	yes	49:11	labour
32	ROP	reg.	0.050	F	yes	31:00	labour
33	ROP	reg.	0.047	F	yes	41:00	labour
34	ROP	reg.	0.040	F	yes	19:00	labour
35	ROP	reg.	0.030	F	yes	35:00	labour
36	LSA	reg.	0.032	F	yes	22:20	labour
37	RSA	reg.	0.030	F	yes	9:36	labour
38	RSA	reg.	0.035	F	nil	99:55	inertia
39	LOA	reg.	0.015	F	nil	25:00	inertia
40	ROA	reg.	0.014	F	nil	36:28	inertia
41	ROA	irreg.	0.012	F	nil	44:00	inertia
42	LOP	irreg.	0.068	M	nil	86:00	inertia
43	ROP	reg.	0.018	F	nil	58:00	inertia
44	ROP	irreg.	—	L	nil	40:24	inertia
45	LOA	reg.	0.080	F	nil	72:00	prodromal
46	LOA	reg.	0.100	F	nil	36:00	false
47	LOA	irreg.	0.100	F	nil	30:00	false
48	LOA	reg.	0.080	F M	nil	18:00	false
49	LOA	irreg.	0.080	F	nil	18:10	false
50	—	irreg.	—	—	nil	—	Braxton-Hicks
51	ROP	irreg.	—	F M	yes	21:00	labour
52	LOA	irreg.	—	—	nil	20:25	not in labour

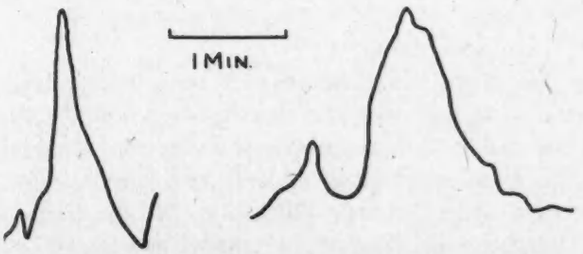

Fig. 6.—Record 49, shown on the left, is a typical fundal contraction during false labour of a patient 4 days before the onset of true labour. Record 7, shown on the right, is from the same area of the same patient 12 hours after true labour began. Note the decreased rate of contraction of the true labour contraction.

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Record 42, Fig. 7, shows a rate of 0.066 inches per second which alone would be considered in the normal labour group. However, the mid-zone was dominant and there was considerable activity in the lower segment with inco-ordinate action between the three zones from which records were made. Using the classification of disordered action of the uterus as set forth by Jeffcoat[16] this case would be considered a colicky uterus and not true primary uterine inertia.

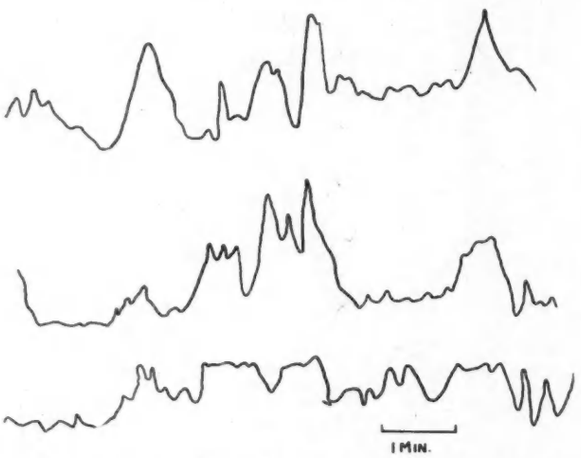

Fig. 7.—A typical tracing of a "colicky uterus". The upper record is from the fundus, the middle record is from the mid-zone and the lower record is from the lower segment, all in the mid line.

The patient from which record number 44 was made had continuous pain, dominance in the lower segment and minimal unmeasurable contractions in the fundus and mid-zone. The uterus, clinically, was hypertonic. This could be classified as a hyperirritable lower segment after Reynolds,[27] although this does not explain why it is hyperirritable.

Records 19 and 40, Fig. 8, were taken from the same patient. The first tracing, number 19, was taken 25 hours before delivery and showed the characteristics of an efficient labour. Nine hours later further dilatation of the cervix stopped and the patient complained of severe pain. A further tracing (number 40) was taken, and this gave the typical primary uterine inertia pattern, low frequency, low amplitude, lack of fundal dominance, plus a slow rate of rise in the fundus.

Records 22 and 41 are from the same patient. The clinical history here was similar to that described in the last paragraph, although in this case x-rays showed same cephalo-pelvic disproportion and a Cæsarean section was done.

Tracing number 44 was taken 25 hours before delivery. There was some slight activity in the lower segment but the mid-zone and fundus showed no measurable activity. The patient eventually delivered spontaneously. Record number 51 was from a somewhat similar case except that the cervix continued to dilate throughout labour. The tracing was made 3 hours before delivery. This case was considered to be labour clinically, since the patient progressed steadily toward delivery.

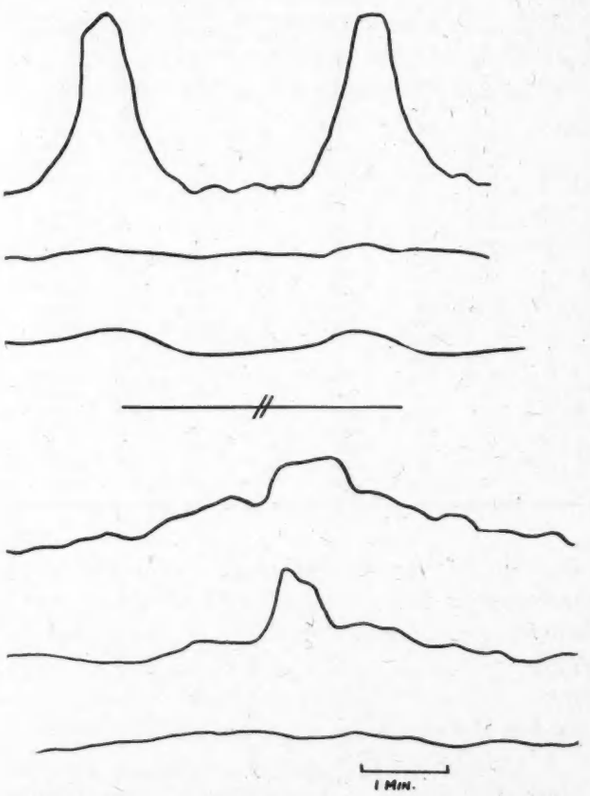

Fig. 8.—The upper three records are from a patient in good labour. Reading from the top down the records are from the: fundus, mid-zone and lower segment. The lower records are from the same patient 9 hours later showing the development of primary uterine inertia. The records from the top down are: fundus, mid-zone and lower segment.

The TKG of record number 50 was from a patient with triplets in the seventh month of gestation. At the time of the tracing only the occasional Braxton Hicks contraction was recorded.[15]

CONCLUSIONS

From this small series of cases it seems that the rate of contraction in the fundus is a valuable adjunct to the other factors measured in assessing a labour pattern as shown by the TKD. No one factor in itself is diagnostic of an efficient labour, but if the rate, rhythm, dominance and

co-ordination are within normal limits then labour can be viewed without concern.

The TKD forms a very useful supplement to clinical impressions, especially in borderline cases of abnormal labours since objective measurements can be made on the TKG's.

At the present stage of knowledge it is not possible to predict the expected length of labour but it is possible to show the probable type of labour. Knowing the type of labour a patient will have, appropriate steps can be taken in cases of primary uterine inertia to institute early treatment or in cases of false labour to leave the patient alone and not subject her to unnecessary procedures or expense.

SUMMARY

1. There appears to be an optimal rate of contraction of the uterus at which parturition can be accomplished most easily.

2. The rate of rise of the fundal contractions of the uterus, calculated from tracings made with a tokodynamometer by dividing the height of fundal contractions, in inches, by the contracting time, in seconds, of the uterus, is a valuable calculation to assist in classifying various types of labour.

3. False labour has a rate of rise approximately three times that of normal labour.

4. Primary uterine inertia has a rate of rise approximately one-third that of normal labour.

III. TKD TRACINGS OF VARIATIONS IN WATER PRESSURE IN LATEX MODEL OF FULL TERM UTERUS

In recent years workers interested in the physiology of the uterus during labour have been recording uterine action by various devices. have fallen into two general types: those measuring electrical potentials at various places on the uterus, and those utilizing some mechanical means of recording changes during a contraction. In 1948 Dill and Maiden[10] devised an instrument for measuring electrical potentials, but they were pessimistic about this method of investigation because of technical difficulties. Steer and Herscht[32] took somewhat the same view. In 1952 Halliday and Heyns[12] produced an instrument with which they have recorded changes in electrical potential during labour with considerable success.

The chief exponents of the mechanical instruments for recorders have been Lorand,[17] Murphy[21] and Reynolds.[29] Murphy[20] used a tocograph similar to the one used by Lorand in his studies in 1937. This method of recording has been refined by Reynolds and his co-workers[26] by using the three or more electronically recording strain gauges which measure the change in curvature of the abdomen overlying the anterior wall of the uterus. Caldeyro and Alvarez[7] have used the TKD in conjunction with manometric recordings of the amniotic pressure during labour.

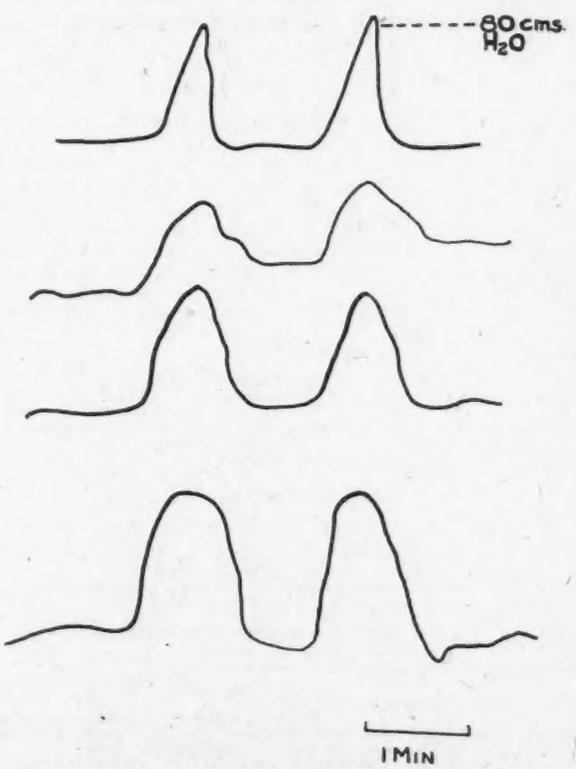

Fig. 9.—Typical records made from the model uterus without the effect of the abdominal wall. The records from the top down are: manometer reading, strain gauges on the fundus, mid-zone and lower segment. Note the reversed gradient.

There is some conflict in the results obtained by these two basic methods of recording. The tokodynamometer shows a definite gradient of activity during a contraction, with the maximum excursion shown in the fundus and the least in the lower segment. This finding was correlated by Reynolds[28] with the fact that there is more muscle tissue in the fundal portion of the uterus, there is greater amount of actomyosin in this area, and, on a mechanical basis, the radius of curvature is greater in the upper segment. All of these factors, in Reynolds' view, point to the conclusion that there is greater muscle activity and force in the fundus which dilates the cervix

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and pushes the baby through the birth canal. This reasoning has long been accepted, as it seemed to be "commonsense" and at the same time agreed with most observations.

Halliday and Heyns published their preliminary observations with their method of recording electrical potentials.[12] They found that during the first stage of labour the electrical potentials were of the same voltage and at the same point in time in both the fundus and the lower segment of the uterus. From these observations they advance the hypothesis that the uterus during the first stage of labour is contracting with equal activity all over its surface. From this premise they have devised another theory to explain the dilatation of the cervix by likening it to two balloons, one inside another, the outer balloon having a hole in the end. These same workers criticize Reynolds' work on the grounds that the uterus rotates about its cervical attachments so that the fundal portion is thrust up toward the anterior abdominal wall, hence reducing the radius of curvature more than in the lower segment, and thus producing an apparent gradient of activity. They feel also that the elasticity of the tissue of the abdominal wall between the gauge and the uterus influences the record. In summary, they conclude that the strain gauge recording is the end result of a chain of events with no simple relationship between them.

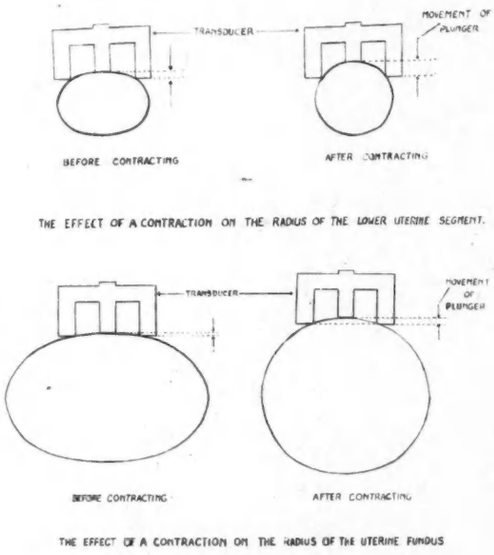


Fig. 10

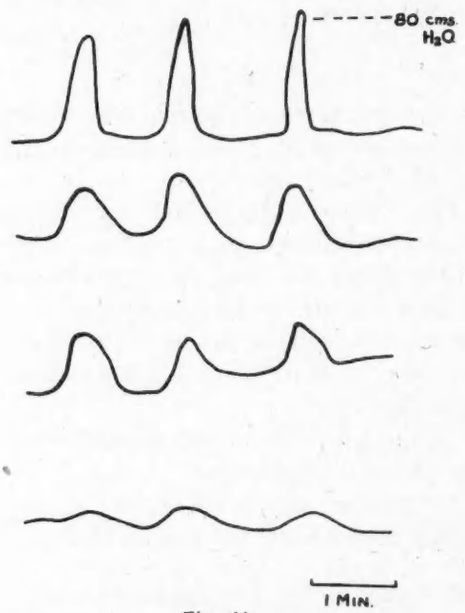


Fig. 11

Fig. 11.—Typical records made from the model uterus with the effect of the abdominal wall. The records from the top down are: manometer reading, strain gauges on the fundus, mid-zone and lower segment.

After the results of Halliday and Heyns were published it was decided to try to devise some sort of model to reproduce the results observed with a TKD during labour.

Method.—A life-size latex model of a gravid uterus was constructed. The dimensions of the uterus were obtained from the life-size pictures of Dickenson.[9] To construct the model, a plasticine positive model was sculptured. From this a plaster cast was made in two halves. Hot parawax was poured into the plaster negative mold, allowed to stand for a few minutes, and then the remaining molten wax was allowed to drain out. The result was a thin-walled wax positive model of the gravid uterus. This wax model was affixed to a stand and painted with liquid latex in successive layers with several layers of cheese cloth impregnated in the latex so that the finished model would not stretch when pressure was applied.

When the latex and cloth had been built up to sufficient thickness, the model was struck a sharp blow, breaking the wax positive under the latex, and the bits of broken wax were pulled out through the "cervix" of the latex model.

The model was then connected with a water manometer and source of water pressure, and a device for recording the water (amniotic) pressure on the same chart as the three strain gauges of the TKD was set up. The posterior half of the plaster cast was used as a bed for the latex uterus to simulate the pelvic bed. The lower portion of the anterior half of the plaster cast was cut off and placed over the anterior lower portion of the model to simulate the anterior pelvic tissues. Thicknesses of towelling were used to simulate the anterior abdominal wall. The three strain gauges were placed in the midline at the fundus and lower segment, corresponding to areas B, E and H of Reynolds.[26]

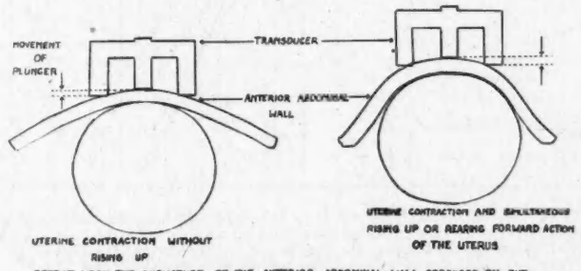
With the model uterus set up as described, the water pressure was varied in such a manner as to produce tracings similar to those recorded by Caldeyro and Alvarez with a manometer connected to the amniotic sac during labour.

RESULTS

Repeated tracings made with this apparatus can be seen in the examples shown in Fig. 9. It will be observed that the gradient is in the opposite direction from that seen in tracings of normal labour, the maximum excursion being seen here in the lower segment, with the fundus showing the least.

This result would be expected from a theoretical consideration of the situation and is shown by the diagrams of Fig. 10. As pressure is increased the uterus becomes more spheroidal and less of an oblate spheroid in cross section. Hence the change in curvature is greater in the lower segment than in the fundus, and therefore the plunger of the strain gauge on the lower segment is pushed inward the furthest.

If the model uterus is made to simulate the motion of rotation about the cervix with the rising of the fundus pushing against the abdominal wall, and records are then taken, the gradient as observed during labour is reproduced, as the examples in Fig. 11 illustrate. The action of the abdominal wall was simulated by fastening the towelling representing the abdominal wall to the plaster bed and then apply-



EFFECT UPON THE CURVATURE OF THE ANTERIOR ABDOMINAL WALL PRODUCED BY THE RISING-UP ACTION OF THE UTERINE FUNDUS DURING A CONTRACTION.

Fig. 12

ing the pressure. If at the same time the uterus was raised by a sling an even greater excursion could be obtained than those shown.

The effect of the rising-up action of the uterus can be shown diagrammatically in Fig. 12. Here it is readily seen that the restriction of the abdominal wall to the rising uterus decreased the radius of the fundus more than if this rising up did not exist.

The rotation of the uterine body about its pelvic attachments is ascribed in the standard textbooks to the contraction of the round ligaments[11] but it does not seem reasonable that a structure with such a small amount of contractile tissue working at such an inefficient angle could produce this action. It is felt that the rotation is produced by the splinting action of the fetus *in utero*. As the uterus contracts with the head engaged (and therefore fixed), the fundal portion of the uterus rises up because the axis of the baby's body swings in line with the upward projection of the axis of the pelvic curve of Carus along which the baby must pass.

CONCLUSIONS

From these experiments with a latex model of a gravid uterus, it would seem that the records of the tokodynamometer are really tracings of the change in curvature of the abdominal wall produced by the contraction of the uterus plus the rotation of the uterus about its pelvic attachments by the splinting action of the fetus *in utero*.

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INTRANASAL CORTICOTROPIN—ITS PHYSIOLOGICAL AND CLINICAL EFFECTS*

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HERETOFORE corticotropin has been found to be effective only when administered parenterally. ACTH given orally,[1] or as an aerosol spray has proved inactive.[2] However, from treating cases of diabetes insipidus it is known that posterior pituitary extracts although protein in nature are absorbed through the nasal mucosa.

Paulson[3] and his associates were the first to demonstrate that ACTH likewise could be absorbed through the nasal mucosa provided a suitable preparation was used. The specifications of such a preparation included: (a) molecular weight small enough to penetrate the intact mucosa; (b) a highly purified type of ACTH which would not cause irritation and sneezing; (c) avoidance of other powder constituents which would interfere with absorption; and (d) a particle size which would adhere to the nasal mucosa. With such a preparation administered intranasally Paulson was able to demonstrate a lowering of the circulating eosinophils and with larger doses of 30 to 50 units daily induced remission in rheumatoid arthritis.

The obvious advantages of such a simple means of administering ACTH prompted further investigation of this route using a specially prepared highly purified corticotropin.** Its effectiveness was measured by blood eosinophil responses and urinary 17-ketosteroid excretion in normal individuals and by clinical observations in a variety of conditions known to respond to ACTH or cortisone.

PHYSIOLOGICAL EFFECTS OF INTRANASAL CORTICOTROPIN

(a) *Circulating eosinophils.*—For the purpose of this investigation 9 normal individuals were chosen and while fasting (9 a.m. to 1 p.m.) their eosinophil counts determined immediately before and 4 hours after 40 mgm. of intranasal placebo. It will be seen from the accompanying Fig. 1 that in only one individual (C), the eosinophil count was reduced by 50% of its pretreatment

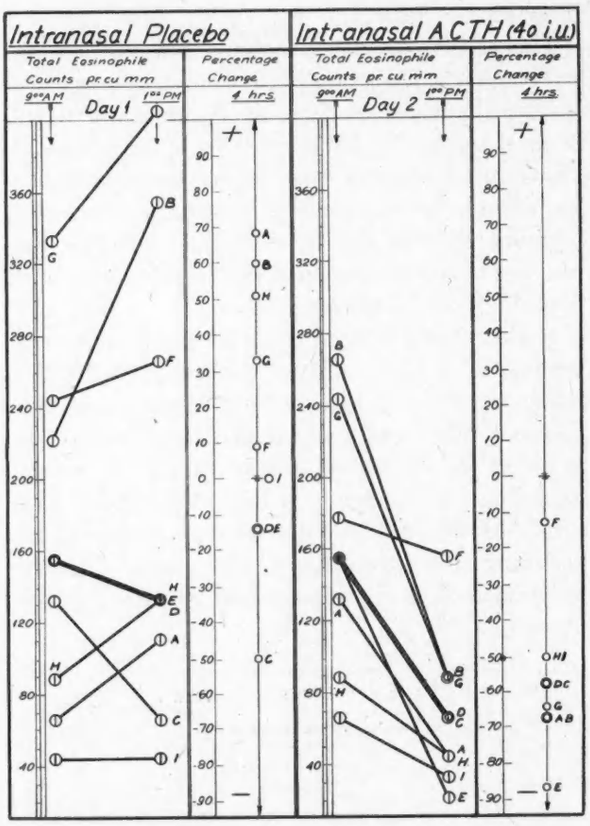

Fig. 1.—Comparison of 4 hour eosinophil response to administration of intranasal placebo and intranasal ACTH (40 I.U.) on consecutive days, in nine normal subjects. Preparations were administered at 9 a.m. on both days.

level, and in the other 2 (D and E) the eosinophil drop was not very significant—14.2% in each. In one (I) there was no change. In subjects B, A, G and H the eosinophil count increased by 59.9; 68.2; 33.3; and 51.1% respectively.

The following day the same individuals were given 40 I.U. of ACTH in a single intranasal dose. Their eosinophil counts were determined before and four hours after the intranasal ACTH. It will be observed from Fig. 1, that in eight out of nine individuals, the eosinophil counts were lowered by 50% or more: Subject 1, 52%; D, 57.4%; B, 66.9%; A, 66.9%; C, 57.4%; G, 63.9%; E, 85.8%; H and I, 50.0%, and in F the eosinophil count fell by only 12.9%.

In yet another investigation, nine normal individuals were given 40 I.U. of ACTH by the

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intranasal route. Eosinophil counts were done at two-hourly intervals for six hours to determine the rate of absorption of ACTH from the nasal mucosa. It would appear from Fig. 2 that the maximum eosinopenia response after intranasal ACTH occurs at about the same time as that which follows ACTH intramuscular injection. The computed average decrease in the eosinophil counts for the nine normal subjects in this study show it to lie well below 50% (Fig. 2X).

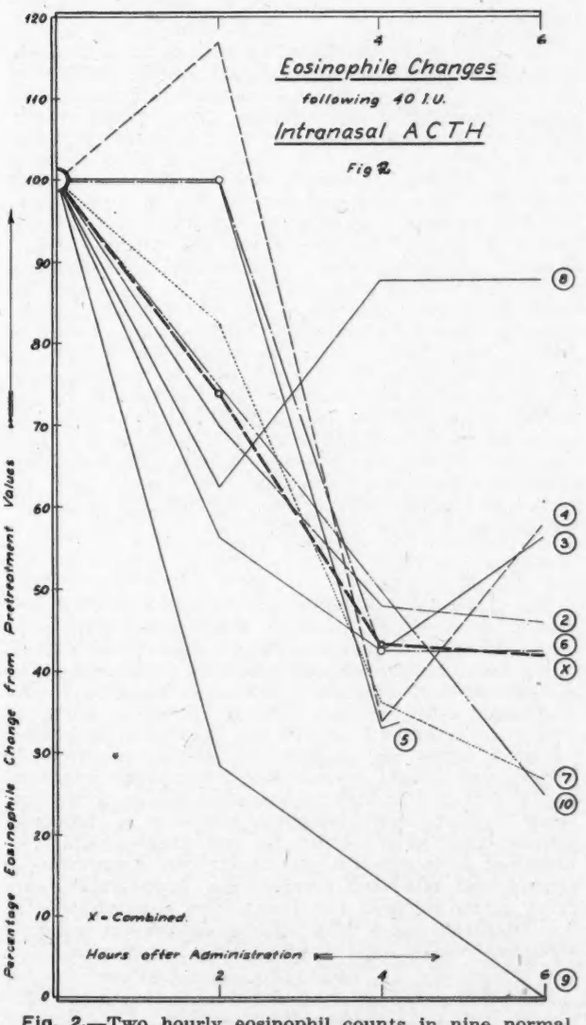

Fig. 2.—Two hourly eosinophil counts in nine normal subjects after (40 I.U.) ACTH—intranasally.

Studies, yet incomplete, indicate that the minimum dose of intranasal corticotropin which will depress circulating eosinophils by 50% or more, ranges from 20 to 40 I.U.

(b) *Urinary 17-ketosteroid excretion.*—It will be seen from the accompanying Fig. 3, that intranasal ACTH has markedly influenced urinary 17-ketosteroids excretion in nine normal subjects, and that no such changes were observed in the same individuals when previously given the intranasal placebo.

On the first day, the nine normal subjects were given mgm. of placebo intranasally and their urine collected for the next six hours (8 a.m. to 2 p.m.) for 17-ketosteroids determination. The results are presented in Fig. 3. On the following day, the same individuals were given 40 units of intranasal ACTH in a single dose and urine was again collected for 6 hours for ketosteroids determination.

It will be seen from Fig. 3, that following 40 units of intranasal corticotropin, there was an increase in urinary 17-ketosteroids excretion in all subjects as compared with the day before when they had taken placebo only.

In subject A, the urinary 17 ketosteroids—6 hours increased by 1.5 mgm., 68.2%; B 1.8 mgm., 58.1%; C 4.2 mgm., 87.5%; D 9.3 mgm., 152.4%; E 3.6 mgm., 83.7%; F 9.1 mgm., 178.4%; G 8.5 mgm., 212.5%; H 0.9 mgm., 22.0%; I 2.0 mgm., 39.2%.

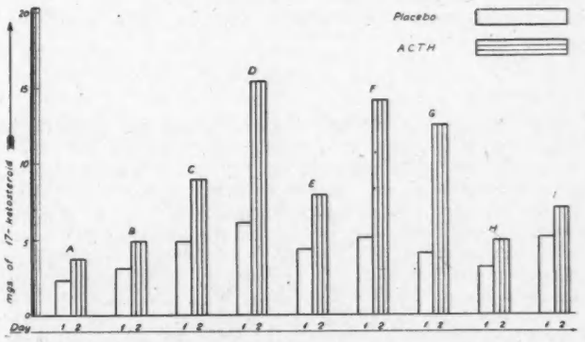

Fig. 3.—17-ketosteroid excretion measured on consecutive days after administration of intranasal placebo and 40 I.U. intranasal ACTH. Nine normal subjects.

Thus it will be seen from the above findings that a suitably prepared ACTH preparation when given to normal individuals in therapeutic doses can be absorbed from the nasal mucosa, lower the circulating eosinophils by more than 50% (in the majority of cases) and produce a considerable increase in urinary 17-ketosteroids.

In view of these findings it was decided to continue this investigation and observe the effect of the intranasal preparation in various clinical disorders known to respond to ACTH therapy.

Therapeutic trials with intranasal corticotropin.—In all, 17 consecutive patients with conditions known to respond to ACTH or cortisone were treated. Summary of the results is found in

Table I and brief case reports are presented below:

TABLE I.

A TABLE OF CLINICAL CONDITIONS TREATED WITH INTRANASAL CORTICOTROPIN

Clinical condition	*No.*	*Improvement*	*No improvement*
Nephrosis	1	1	—
Gouty arthritis	4	4	—
Rheumatoid arthritis	5	3	2
Bronchial asthma	4	3	1
Allergic rhinitis	1	1	—
Sydenham's chorea	1	1	—
Toxic delirium	1	1	—

CASE REPORTS

CASE 1

L.S. A three year old boy had nephrosis for sixteen months. He had excellent results following treatment with intramuscular ACTH on three separate occasions. When severe œdema again necessitated treatment intranasal ACTH, 40 I.U. was administered by insufflation, twice daily for eight days. Two days after profuse diuresis began and body weight dropped from 48 pounds to 38½ pounds. When discharged from hospital on the fourteenth day the weight was 31 pounds. The clearing of œdema which followed intranasal ACTH was at least as complete as that previously obtained from parenteral ACTH.

CASE 2

P.C. A 70 year old man was admitted for cystoscopy following which he developed acute gout in the right first metatarsal-phalangeal joint. He had a history of two previous attacks of gout occurring under identical circumstances which subsided gradually over several weeks. Treatment was begun on the 4th day with 20 I.U. of intranasal ACTH every six hours. Within 24 hours, there was virtually complete subsidence of all signs and symptoms. The dose was decreased to 20 units every 12 hours for one day and 10 units every 12 hours for three days and then discontinued. The clinical remission persisted following cessation of therapy.

CASE 3

M.G. Age 59. This patient had a history of two prolonged and severe attacks of asthma within the past year. On both occasions hospitalization was necessary, and the symptoms lasted several weeks each time. She was re-admitted to hospital with another exacerbation and only temporary and inadequate relief was obtained from repeated use of adrenalin and aminophylline. On the 11th day of hospitalization intranasal ACTH was begun in a dosage of 20 units every 12 hours. Considerable relief was experienced within a few hours of the first dose and symptoms almost completely cleared in 12 hours. The dose of intranasal ACTH was reduced to 10 units every 12 hours on the sixth day and continued for a total of 14 days. Thereafter she remained free of symptoms.

CASE 4

Mr. A.L. Age 49. Severe rheumatoid arthritis involving knees, hands, wrists and right shoulder. He was first given 40 units of ACTH by intramuscular injection each day for 6 days. Pain entirely disappeared and swelling decreased. Some increased range of movement was noted and he was euphoric. He slept and ate better. When ACTH was withdrawn a severe exacerbation occurred. A week later 20 units of intranasal ACTH was given twice daily for 6 days without producing any appreciable effect on symptoms or signs.

CASE 5

Mr. K.A. Age 22. Extremely severe rheumatoid arthritis with spondylitis and wasting. There was subluxation of right knee, marked muscular wasting and involvement of nearly every joint. He received 40 units of ACTH intramuscularly for six days and enjoyed improved well being and some increased mobility of joints. After withdrawal of ACTH, return of the signs to pretreatment state occurred. Intranasal ACTH 20 units twice daily failed to produce any improvement in this case.

CASE 6

Mrs. J.D. Age 50. This patient had been virtually incapacitated through habituation to various drugs over the past several years. She was admitted to hospital, confused, disorientated and vomiting after taking twenty Sedormid tablets. A diagnosis of toxic delirium was made. She remained in this state for several days showing extensive agitation, a gross tremor and pulse rate of about 120. She complained bitterly of pain in her back, limbs and asked repeatedly for barbiturates and analgesics. She did not improve on therapy with intravenous dextrose, saline and water-soluble vitamins. She was then given intranasal ACTH, 40 I.U. every 12 hours with the result that within 12 hours of the first dose she became mentally more clear and alert, the pulse rate returned to normal, and there was considerable relief of pains in the back and limbs. After the second day she no longer requested analgesics and stated that for the first time in weeks she was virtually free of pains in the muscles. The intranasal ACTH was discontinued abruptly after several days and her former symptoms of agitation, tremulousness, disorientation, tachycardia and muscle pain recurred. Intranasal ACTH was administered again and satisfactory remission produced.

CASE 7

Mrs. L.C. Age 65, had a severe attack of bronchial asthma in April, 1953, which was treated with intranasal ACTH, 40 units every eight hours. Within 3 hours of the first dose considerable relief was noted and within 8 hours almost complete relief was experienced. She continued taking 40 I.U. daily for 3 weeks when the supply of intranasal ACTH ran out. Two days later wheezing began and 3 days later she was experiencing very severe asthma again. After a few days, intranasal ACTH was given to her once more in a dose of 20 units every 8 hours, with complete remission of bronchial asthma. One month later she had another attack of bronchial asthma which was once more brought under control with intranasal corticotropin; considerable relief being noted within a few hours. The intranasal ACTH was continued for 8 days and the patient has remained symptom-free during the subsequent two months.

CASE 8

L.N. A boy aged 16 with severe chorea of five days' duration and with a previous history of Sydenham's chorea successfully treated with cortisone, was given intranasal ACTH, 40 units three times a day for one day, 40 units twice a day for two days and 40 units daily for 1 week. On the third day, the choreiform movements were considerably diminished in frequency and in force and on the sixth day the only remaining sign was a slight tremor of the tongue. On the seventh day the intranasal ACTH was discontinued with no recurrence of choreiform movements.

CASE 9

C.L. A 54 year old veteran with a history of recurrent gout was admitted to hospital with acute swelling, redness and pain in left wrist and hand which had been

present for several days. Serum acid was 6.6 mgm. %. He was given intranasal ACTH 40 units every 8 hours for two days. Six hours after the first dose he noted considerable relief but there was no objective change. The following morning the swelling and redness had subsided considerably and there was very little pain. The second day there was complete subjective and objective remission. He was maintained symptom-free for five days before exhaustion of supplies of intranasal ACTH necessitated use of ACTH by injection during the period of tapering off of therapy.

CASE 10

Mr. K.L. Age 49. He was admitted to hospital with recurring gouty arthritis involving the left wrist, knee and ankle. Serum uric acid 9.9 mgm. %. He was given intranasal ACTH 20 units every eight hours and in thirty hours there was almost complete disappearance of swelling and redness. The supply of intranasal ACTH ran out and symptoms and signs recurred in about twenty-four hours. When intranasal ACTH was resumed in doses of 20 units every 8 hours for two days, followed by 20 units every 12 hours prompt remission was again induced.

CASE 11

Mrs. J.M. Rheumatoid arthritis of three years' duration with an acute exacerbation. Swelling of hands, ankles, feet and knees made walking difficult and she had to be assisted out of bed and helped to dress. The first dose of 20 units of intranasal ACTH taken at 10 p.m. resulted in her being able to get out of bed unassisted the following morning. Intranasal ACTH was continued in doses of 20 units three times daily for two days and thereafter 20 units twice a day or once a day for several weeks. By the third day of treatment she was virtually free of pains and able to perform her household duties in a normal manner. Satisfactory remission continued as long as she was taking 20 units or more per day of intranasal ACTH. An attempt to maintain remission on 5 units per day resulted in return of many symptoms. When the supply of intranasal ACTH ran out she suffered a gradual exacerbation. On another occasion her symptoms worsened steadily during six day trial with different intranasal corticotropin. On the first day after restarting the original type of intranasal ACTH another remission began. The patient is still continuing on small doses averaging about 10 units per day and enjoys considerable, though not complete, remission of her symptoms.

CASE 12

Mrs. M.A., 65 year old housewife with a chronic tophaceous gout of 25 years' duration. She had been virtually bedridden for the past year and had massive tophi on hands, feet, knees, elbows and smaller tophi in ears and bridge of the nose. She was suffering a great deal because of destruction of bone particularly in the region of the left hip joint. The first dose of 15 units of intranasal ACTH was taken at 9 p.m. and the next morning there was appreciable lessening of pain and increased range of movements in most joints. The clinical improvement was surprising and she stated that the relief of pain made her feel as though she "were in Heaven". The therapy was continued for about three weeks and tapered off after which relapse occurred and again ACTH was administered intranasally and again remission was induced to the point where she was able to get out of bed, use a wheel chair and go riding in the car. Concomitantly a persistent stuffiness of the nose disappeared.

CASE 13

A 38 year old veteran had suffered almost continuously with asthma since a prolonged bout of pneumonia in 1942. Previous efforts to induce remission with cortisone had failed and he was using adrenalin several times per day by injection. Intranasal ACTH was given in doses of 40 units three times daily for three days without changing the clinical picture. Thereafter a similar dose of ACTH intramuscularly was continued but likewise had no effect on the asthma.

CASE 14

Mrs. I.B. A 43 year old housewife with a history of asthma for more than 10 years. At the time of the clinical trial the asthma was severe in spite of the liberal use of parenteral adrenalin and aminophylline suppositories. ACTH intranasally in a dose of 40 units twice daily induced a satisfactory remission and on the second day of treatment the patient noted that "breathing is wonderful" and "for the first time in months can get a good breath". Intranasal ACTH was used intermittently for the next few months and in this case it was found that less than 15 units per day was ineffective.

CASE 15

Mrs. W.G., a 56 year old patient with a 16 year history of rheumatoid arthritis particularly severe in knees and hips. 24 hours after starting intranasal ACTH 40 units daily she noted considerable improvement and within a few days walked without a limp which had been present for years. She had been confined to the house for two years by arthritic symptoms but now is able to go out with only slight difficulty.

CASE 16

Mrs. D.S., 68 years old. Chronic rheumatoid arthritis involving left hip, knee and foot with much difficulty in walking accustomed to using two canes. Three years ago responded to ACTH but showed no improvement while treated with cortisone. Intranasal ACTH, 20 units three times daily induced a considerable improvement the following day. After one week the dosage was reduced to 40 units daily and two weeks later to 20 units a day. Ten units were tried but the pain recurred especially in the left leg. After two months of treatment she is feeling better than she had for years. Coincidentally a severe conjunctivitis present for five months has cleared up during the ACTH therapy.

CASE 17

Mrs. C. de M.T., age 38. Allergic rhinitis and hay fever of eight years' duration with no response to antihistamines or desensitization procedures. Intranasal ACTH started with 20 units three times daily for two days, 20 units twice daily for two days and 20 units once a day thereafter. On the first day of treatment the stuffy feeling in the head cleared, nasal secretions diminished and the patient could breathe freely and for the first time in six years she could taste her food. On the second day of treatment she could breathe perfectly well, her head felt clearer than it had for years and was not affected by visit to the country which usually made her feel much worse. She is free of symptoms now taking 20 units of intranasal ACTH every three or four days and the turgid nasal mucosa and polypi which were obstructing her nasal passage have disappeared.

COMMENTS

1. It appears that a suitably prepared and formulated ACTH preparation can be effectively absorbed through the nasal mucosa.

2. Its effectiveness has been demonstrated in a group of normal individuals in whom a satisfactory decrease in circulating eosinophils and increase in urinary 17-ketosteroids followed a single dose of 40 I.U. intranasal corticotropin.

3. Definite clinical improvement was observed in 14 of 17 consecutive patients treated with the

intranasal corticotropin preparation. This improvement was satisfactorily maintained as long as they continued to take adequate doses of the intranasally administered hormone. One of the three patients who failed to improve did not respond to intramuscular ACTH either.

4. No untoward manifestations were observed apart from slight nasal irritation and occasional sneezing in the first few minutes after taking intranasal corticotropin.

ADDENDUM

Since the submission of this report, an additional 44 cases were treated with intranasal corticotropin for periods varying from several months up to one year (Case 11). In only 2 cases some allergic phenomena were encountered. The effective therapeutic dose ranges from 60 to 80 units, and the lowest maintenance dose 10 units per day.

We are grateful to Dr. J. Feller, Ottawa, for including two of his patients in this series.

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RÉSUMÉ

1. Il semble que certaines préparations d'ACTH puissent être absorbées par la muqueuse nasale de façon satisfaisante.

2. Comme preuve à l'appui, un groupe de sujets normaux présentèrent une diminution des éosinophiles dans leur circulation périphérique ainsi qu'une augmentation du taux d'excrétion dans l'urine des 17-cétostéroïdes après une seule application intranasale de 40 u.i. de corticotropine.

3. De 17 malades traités à la corticotropine intranasale, 14 montrèrent des signes d'amélioration clinique très nette. Cette amélioration persista aussi longtemps qu'ils continuèrent à prendre des doses suffisantes d'hormone par voie intranasale. Un des trois malades réfractaires au traitement n'eut pas de meilleur résultat quand il recut l'ACTH par injections intramusculaires.

4. A part une légère irritation du nez et quelques éternuements tôt après l'instillation, aucun effet fâcheux ne suivit l'administration intranasale de corticotropine.

M.R.D.

ADIPOSITY AND ATHEROSCLEROSIS*

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FOR LONG, the medical profession has held the view that adiposity is undesirable, and that those who habitually overeat are likely to die prematurely from some form of cardiovascular disease. Recent studies from actuarial data have forcefully confirmed this impression, and have assigned to the degenerative cardiovascular disease the major rôle in the excessive mortality seen in the overweight members of the population. Indeed, these studies have indicated that most morbid processes are adversely affected by adiposity. Clearly, this effect is often nonspecific, the addition as it were, of insult to injury. Sometimes, however, the influence of adiposity appears strong enough to suggest that it may be playing a more important rôle. Since atherosclerosis is the underlying basis for the greater part of degenerative cardiovascular disease, it is worth while examining the evidence on which adiposity is held to play a part in its complex pathogenesis.

Until recently, atherosclerosis was considered an inevitable consequence of ageing. Today our approach to its etiology is more hopeful and most would agree that the basic cause of this morbid process may well be a metabolic defect. What this defect is, we cannot say, but we may assume that it involves the biosynthesis, the transport, the catabolism *or* the excretion of certain lipids and, perhaps, especially of cholesterol, in the form of lipoproteins. Many methods have been used to study the blood lipids. All show considerable deviations from the normal plasma lipid pattern, which is especially marked in the younger victims of atherosclerosis. This is so, whether they have a condition known to affect lipid metabolism, like diabetes, or whether they have an inborne error of metabolism, which sees its most marked expression in cases of familial xanthomatosis. If it be agreed that severe atherosclerosis in young subjects is the result of a disorder of lipid metabolism, we must then ask ourselves whether we may similarly ascribe atherosclerosis in the older population to what must be a most widespread disorder. If we *do* accept such a thesis, we must presume the metabolic derangement generally speaking to be of a low order, so that its effects become apparent, as a rule, rather late in life. If this is so, we may expect to find factors which would have an accelerating influence on such a disorder. Adiposity has been accused of such a rôle.

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Just what constitutes adiposity, and how common is it? There is, of course, no sharp line dividing the overweight from the normal person. Most actuarial statisticians have felt the best criterion to be an arbitrary percentage departure from average weight-for-height. The Metropolitan Life, for example, has prepared a set of standards based on the average weight of healthy persons between 25 and 30 years of age, making due allowance for those factors in body structure known to influence weight. These are then presented as tables of "ideal weight" for men or women of small, medium or large build. Overweight is then defined by a deviation of 10% or more from these standards; about 20% of the American population belong to this class. Marked adiposity, so-called pathological obesity, is spoken of when the weight departs from the ideal by more than 25%; one-third of the overweight, or about one person in every fifteen, may be so classified.

Even the best life insurance statistics can hardly be expected to give us the answer to our precise problem. However, the problem is set in proper perspective by looking at one recent study by the Metropolitan Life group.[1] They did a 25-year follow-up on 50,000 insured persons, aged 20 to 64 when insured—all of whom were charged extra premiums merely by reason of overweight. In the whole group, the mortality was considerably higher than for "standard risks" and the disparity was especially marked in the younger group. That is, those who were 20 to 29 when examined for insurance had a mortality rate which was 80% greater than standard expectations, while in the group aged 51 to 64 on examination, the deaths were 31% greater than expected. Again, the authors stress that degenerative diseases of the heart, arteries and kidneys contributed most of this excess mortality. There are of course several ways in which adiposity might contribute to this result. Witness a number of studies on various industrial groups[2] which correlate significantly overweight and the development and progression of arterial hypertension. Then, there is the well-known adverse effect of overnutrition on the incidence and course of diabetes, which has been forcibly reaffirmed in all recent studies. It is these complex interrelations, as well as the gross inaccuracies inherent in all data obtained from death certificates, that make other methods of study essential in this problem.

Of much interest are the various detailed analyses which have been made of autopsy material. Wilens[3] studied a group of 1,250 necropsies from the Bellevue Hospital, and reported his findings in 1947. He divided his material into three groups, according to the state of nutrition as evaluated by the pathologist at autopsy. Considering only evidence of advanced atherosclerosis, he found that the proportion of the overweight group with this condition was consistently and markedly in excess. For example, at ages 45 to 54, 20% of the adipose subjects—and they were truly adipose—had advanced changes in the arteries, as against 6.7% of those classed as lean; at 65 to 74, the proportions were 45.4% and 20.2% respectively. A special study was made of coronary atherosclerosis, and it too was recorded as being of marked degree more often in the obese and particularly in obese males.

Three years earlier (1944) French and Dock[4] had studied material from the U.S. Army Museum of Pathology and reported on 80 fatal cases of coronary disease in young soldiers (19 to 39). They found a sufficient predominance of adiposity in this group to conclude that "overweight was an important etiological factor in these young men". However, Yater[5] four years later (1948) made a more extensive study of the same material and reached somewhat different conclusions. He reported on 233 coronary deaths —including the group of 80 originally studied. He agreed that the average weight of the group was significantly greater than that of inductees, but pointed out that the same degree of excess weight was present in a further group of 297 men of the same ages who had died of accident. Further, he checked on the weights of the coronary group on induction and found them no higher than average. The only obvious conclusions to be drawn from this study then is that the average American soldier is very well fed and is exposed to what might be termed overnutrition during his army life.

Perhaps another autopsy study might be mentioned, this time from the Mayo Clinic. Ackerman, Dry and Edwards[6] in 1950 reported on 600 autopsies on women. Specifically they studied the hearts from 100 consecutive cases dying in each decade. They found significantly fewer atherosclerotic coronary lesions in those who were reported as underweight by the pathologist. They reported *no more* lesions in the

adipose group than in those of average normal weight. This is actually one of the many pieces of evidence suggesting that underweight may in some way be protective against atherosclerosis while, at the same time, not confirming the thesis that adiposity *per se* is necessarily deleterious.

One war-time study in vital statistics is worthy of notice at this point. Two years ago, Dedichen[7] reviewed mortality statistics from circulatory disease in Oslo for the years 1940 to 1945, when the entire population were under rigid dietary control, and compared them with data for the periods both before and after the period of diminished food intake. There was a striking reduction in mortality which began within a few weeks of the institution of deprivation, reached its lowest point in about three years, and continued with little change until the previous dietary pattern had been restored. Within a short time following the removal of restrictions, the death rate rose again to its pre-deprivation level. This suggests, but does not prove, that the period of comparative undernutrition coincided with a lessened incidence of atherosclerosis, or of its complications. Since one of the important, and often fatal complications is intraluminal thrombosis, it is interesting that Dedichen was also able to demonstrate a striking decrease in thrombo-embolic complications following surgery, during the same deprivation period. Dedichen doesn't claim that his war-time Norwegians actually lost much weight on their admittedly low-fat and low-calorie diets. We may assume that they did lose a considerable amount, but unless all the obese were thereby rendered normal in weight and, unless practically all the circulatory deaths had formerly occurred in this proportion of the population, we could hardly account for the marked drop in mortality by the actual decreased incidence of obesity in the population. I prefer to regard this study as evidence of the value of relative underfeeding in decreasing circulatory deaths, but whether because incidence and progression of atherosclerosis, or because of fewer thrombotic complications. I do not know. Incidentally, Dedichen's findings with reference to thrombo-embolic disease recall many previous reports,[8, 9] mostly in the American literature, dating back to 1927—all of which *have* shown that the really adipose patient is much more liable to such accidents than is the patient of more normal weight.

Moving from statistical studies of populations and autopsy material, to the living subject, we run into many difficulties. not the least is the virtual impossibility of determining before death just who has and who hasn't got atherosclerosis. When such an assessment is attempted on purely clinical grounds, we should realize that anywhere from 30 to 50% of our so-called "normal controls" will have as much or more atherosclerosis than will many of our subjects with clinical symptoms of their disease. Fortunately, most would agree that there is at least some correlation between atherosclerosis and certain plasma lipid abnormalities, although there may be sharp argument about methods of measurement, and the significance to be attached to each! Certainly, where the abnormalities are very gross, as in some cases of diabetes, in nephrosis, and in familial cases of hypercholesterolæmia, then, too, atherosclerosis develops early and progresses rapidly. In such cases, the total cholesterol is greatly elevated and all the more refined methods of lipid study show marked deviation from the normal pattern. One of the best-known but so far least widely used, techniques, is of course that of Gofman and his associates.[10] They have utilized the technique of ultracentrifugal flotation of the serum lipoproteins to identify and measure the various aggregates in which the plasma lipids are transported. Briefly, they feel that they have demonstrated a significant correlation between certain lipo-proteins, designated as Sf 12-20 and 35-100 and the occurrence of marked atherosclerosis. The Sf 35-100 fraction is relatively variable, and has been demonstrated to rise sharply in many persons on high fat intakes. The two together, that is virtually the Sf 12-100 fraction, Gofman regards as "the atherosclerogenic band" of the lipoproteins. More recently[11] Gofman and Jones have found that the Sf 35-100 fraction is significantly elevated in the obese. There is a lesser, though still significant elevation in the Sf 12-20 lipo-proteins. These findings provide yet another argument for weight reduction in the obese.

It will be seen that the case for assigning to adiposity a causative rôle in the development of atherosclerosis is reasonably strong. But I would submit that most of the evidence is open to somewhat different interpretation, which on the whole fits the facts even better. May not the presence of adiposity be in fact merely a very good indicator pointing to habitual over-nutri-

tion? And may we not conclude that it is this over-nutrition which has produced *both* the excess poundage *and* the atherosclerosis? Might not indeed, such habitual over-nutrition be the important factor which in many persons brings to the fore a metabolic derangement of such low order as ordinarily to become apparent only late in life? Obviously, we would not expect it to be the only factor of importance in determining the degree of such a metabolic disorder. There is, in general, an increase in serum lipids and lipo-proteins with advancing age, even in apparently normal subjects, and this increase is particularly apparent after the age of 40, although as Gofman has shown, it becomes of significant degree in males at a considerably earlier age than in females. In addition to such factors as age and sex, it would appear that stress is also of importance in determining significant lipid shifts. For example, Groen[12] and his associates in Amsterdam, using carefully calculated diets, has demonstrated a "reaction pattern" during and following various forms of stress, in certain of their subjects. Whether the stress was mental, physical or was represented by a severe infection, the pattern was one of sharp fall in the serum cholesterol during the stress period. When this period was at an end, and usually after 3 or 4 weeks, there was a rise in cholesterol values, often well above the pre-stress level. Such observations indicate clearly that the controls of lipid metabolism are indeed complex, and many factors, including age, sex, individual constitutional make-up and environmental stress are concerned. This, however, does not vitiate the importance of dietary factors, since after all the diet is one aspect of the patient's environment which we, as physicians, *can* change, provided we are sufficiently impressed with the necessity for so doing.

What is there about the average Western diet which is so inherently bad? Other than its abundance, and ease of procurement, so that most persons eat of it to surfeit, its worst feature is almost certainly its high fat content. Throughout the Western world, where food is not in short supply, more than 40% of total calories are derived from fat. In some countries this fat may be largely vegetable, and the cholesterol content may be relatively low. This apparently makes little difference. We have repeatedly demonstrated, in short term dietary experiments, as have Keys[13] and others, that even on a diet almost completely lacking in cholesterol, the cholesterol content of the blood may rise and the lipid pattern come to show the abnormal features commonly seen in atherosclerotic subjects, provided the fat content and total calories are undesirably high. Conversely, cholesterol may be added in considerable amount to a low fat diet without changing the cholesterol or other lipid levels appreciably.

While there are undoubtedly large differences in individuals in their response to over-nutrition, it is probable that only a very few can tolerate for long the persistent over-eating which leads to obesity without, at the same time, developing some degree of atherosclerosis. It may be that gross over-nutrition, leading to gross adiposity, is quantitatively more atherosclerogenic in a given individual than mild over-nutrition leading to moderate adiposity. Any degree of over-feeding, and particularly of over-feeding with fat, is however clearly undesirable. We have all been looking for some simple test by which we may pick out the potential victim of atherosclerosis so that something may be done about it before it is too late. May we not regard the presence of adiposity as the simplest test of all, and as a certain indication of the need for dietary correction?

When adiposity is to be treated in the comparatively young and otherwise healthy, rapid weight reduction by radical curtailment of calories, as well as of fat, is often the method of choice. In older patients, and particularly when atherosclerosis is clinically evident, we hold that such a course is not only undesirable but hazardous. Some have argued[14] that if this were so, it would be because the lipid abnormalities would be aggravated during the period of rapid utilization of fat stores, and have studied the lipid patterns during such rapid weight reduction. When they found no significant worsening of the pattern, they claimed that such a regimen was therefore safe. But may not the danger lie in an increase in the tendency to thrombotic complications already shown in various studies to be excessive in the adipose group? It is not rare to find that a cerebral or coronary thrombosis in an adipose patient has coincided rather closely with an over-strenuous attempt at weight reduction. For this reason then, I prefer to change the dietary pattern of my adipose atherosclerotics qualitatively, by reducing the fat content sharply, while maintaining the caloric intake at reasonably adequate levels, dropping it only

slowly after some weeks so as to permit of a weight loss of not more than 3 or 4 pounds a month. It is, we believe, safe to carry out rapid reduction in such persons while they are receiving anti-coagulant therapy, following acute atherosclerotic complications, but not otherwise. Some may disagree sharply with these views but all would, I am sure, prefer prophylactic rather than therapeutic weight reduction.

Dr. David Barr[15] has summed up this whole problem very nicely in a recent editorial. He concludes that:

> "During the period while further scientific analyses of the diet are being attempted, a low fat, low calorie diet may be advised with the expectation of partial protection against the rapid development and the complications of atherosclerosis. Since man is an atherosclerotic animal and since the disease probably affects to some degree most individuals in middle or later life, the advice should not be limited to the obese or to those who have suffered obvious complications of atherosclerosis."

This advice will be regarded by some as too radical; certainly it will not be widely followed at this time. I believe that further work will demonstrate its soundness, although it is to be hoped that new discoveries may provide still better means of overcoming the atherosclerogenic tendency provoked by over-nutrition.

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RÉSUMÉ

L'obesité aggrave l'évolution de la plupart des processus morbides. Dans l'état présent de nos connaissances de l'athérome, il semble que cette lésion soit l'effet d'un vice de métabolisme des lipides et des lipoprotides, et que l'adiposité puisse en accélérer le cours. L'obésité simple se définit comme un excédent de 10% ou plus du poids idéal. Si l'excédent dépasse 25%, l'obésité est dite pathologique. D'après les calculs de leurs actuaires, les compagnies d'assurance considèrent les candidats obèses comme de mauvais risques; plus jeune l'obèse, pire le risque. La compilation de nombreux rapports d'autopsies montre que l'athérome se rencontre plus fréquemment chez les sujets obèses qu'autrement. Une communication provenant de la Clinique Mayo, et basée sur une série de 600 autopsies pratiquées sur des femmes, montre que la maigreur peut, d'une certaine manière, protéger contre l'athérome sans toutefois confirmer la thèse voulant que l'obésité soit nécessairement néfaste. Les statistiques démographiques de Norvège semblent indiquer une baisse dans la fréquence de l'athérome et de ses complications pendant les années de sous-alimentation relative, de 1940 à 1945. Les phénomènes thrombo-emboliques post-opératoires furent remarquablement moins nombreux.

Une des principales difficultés à surmonter dans l'étude expérimentale de l'athérome *in vivo* chez les humains, provient de ce que bon nombre de témoins soi-disant normaux peuvent fort bien être athéromateux eux-mêmes. L'importance des lipides du plasma et la méthode de leur détermination sont très discutées. Gofman réussit à isoler, grâce à sa technique d'ultracentrifugation, le groupe de lipides connus depuis sous l'appellation Sf-12 à Sf-100, et impliqués dans l'evolution de l'athérome.

L'obésité suppose une suralimentation habituelle qui peut contribuer au vice de métabolisme mentionné plus haut. A part les facteurs âge et sexe, le "stress" peut occasionner des variations dans la qualité des lipides comme l'indique l'abaissement du taux du cholestérol en période de tension et son retour au niveau initial quand le calme revient. Le pire aspect du régime alimentaire occidental est sa forte teneur en graisse. La suralimentation surtout en graisse est nettement contre-indiquée. Bien qu'une fonte pondérale rapide, obtenue par une restriction sévère en calories, soit relativement sans danger pour les sujets jeunes, de telles mesures ont déja coïncidé avec des thromboses cérébrales ou coronariennes chez de vieux obèses. On doit donc procéder plus lentement avec ces derniers, ou alors leur administrer des anticoagulants. Il est donc préférable que ceux-ci ne perdent pas plus de 3 à 4 livres par mois.

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While experimentation [in the integration of medical teaching] is to be welcomed and encouraged, it should be pointed out that in undertaking experiments schools should be cautious that they lose none of the progress that has been made in placing medical education on a sound educational, scientific, and individual basis. Furthermore it should be realized that it takes time to develop an intelligently designed experiment. To develop a successful experiment a school must first analyze its objectives and resources. If this is done, almost any experiment based on sound educational principles and carried out by a competent and enthusiastic faculty offers promise of serving as a contribution to the improvement of medical education.

It should be kept in mind, however, that it may be extremely difficult to evaluate the results of experiments in medical education. Such evaluation may require continuing observation of their influence on students for several years after graduation.—H. G. Weiskotten, *The Diplomate*, 25: 69, 1953.

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SOME OBSERVATIONS ON CANCER OF THE BREAST*

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A HUNDRED YEARS AGO, when the Faculty of Medicine of Queen's University came into being, Rudolf Virchow (1821-1902), then Professor of Pathological Anatomy at Würzburg, was developing and establishing the "true and fertile doctrine that every morbid structure consists of cells which have been derived from pre-existing cells as a progeny". His book, *Cellularpathologie*, published in Berlin in 1858, influenced medical thought and practice profoundly and a new era in medicine began, especially in the fields of cancer research and treatment.

In 1867, Charles Moore,[1] of the Middlesex Hospital in London, influenced by Virchow and the poor results of local mastectomy for carcinoma of the breast, introduced a more extensive removal of tissues adjacent to the breast. This suggestion was developed by Halstead (1852-1922), who, in 1894[2] reported his experiences of radical mastectomy, the operation which he developed and which has, in the last sixty years, cured more patients with breast cancer than any other method. To it has been added radiotherapy in its various forms and several methods of hormone therapy or hormone withdrawal therapy. The net effect of these medical and surgical measures and of the instruction of the people by organizations such as the Canadian Cancer Society has been to increase the five-year survival rate (cure rate) from about 10% in the first decade of this century to about 40% at the present time, no mean achievement when one recalls that a hundred years ago cancer of the breast, like other forms of cancer, was almost uniformly fatal.

But, although there has thus been much improvement in the last hundred years much still remains to be done to control and cure a disease which still kills 37 out of every 1,000 women who reach maturity (Chase[3]). Re-examination from time to time therefore of the problems involved and of our methods of treatment is desirable.

THE CLINICAL PROBLEM

It is of course obvious that unless the patient seeks advice no treatment can be offered. It is also clear that unless she thinks there is something wrong she is unlikely to seek advice. It follows therefore that the education of the public in the early signs and symptoms of cancer is the cornerstone of the whole edifice of cancer detection and treatment. But, if that cornerstone is to remain in place, the cement of confidence is required, confidence that if she does seek advice she will have a hearing and be examined and that the treatment advised will cure or at least greatly alleviate her ailment.

From the doctor's point of view the diagnostic problem is not always easy. It commonly revolves around the presence of a lump or thickening in the breast or a discharge from the nipple. In late cases when the lump is large and fixed, with distant metastases, no problem exists except that of how best to palliate the condition. But in early cases, where the lump is small, or the breast large, or where fibroadenoepitheliosis (chronic mastitis) is present, diagnosis is frequently impossible without adequate biopsy and histological study. It will be noted that emphasis is placed upon the words adequate biopsy. By adequate biopsy is meant excision of the whole suspected portion of the breast, not merely the parsimonious removal or aspiration of a fragment which may itself be innocent, the malignant tissue escaping removal. Twice recently we have, by great good fortune, and the removal of adequate breast tissue, avoided this tragic error, but at present in our clinic we have two patients dying of their disease in whom the mistake was made elsewhere and a third similar patient committed suicide a year ago.

Of course not all lumps in the breast are malignant. Indeed in women under the age of 30, 99% are simple non-malignant variations on the common theme of fibroadenoepitheliosis and generally a conservative policy may be pursued, especially if the lump is vague in outline, one of many, and more easily felt between the finger and thumb than between the fingers and chest wall. If, on the other hand, it is single, firm, mobile, and enlarging, although probably a simple fibroadenoma, it should be removed, since carcinoma of the breast, though rare before thirty, may occur even at the tender age of ten.[4]

In a woman of 50, however, the situation is vastly different. At her age 85 out of 100 isolated lumps in the breast are malignant and all should be considered cancerous until proved otherwise by adequate biopsy, no matter how simple the

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lesion seems on clinical examination. Indeed it is impossible to overemphasize that in the breast as elsewhere, there are no signs of early cancer which are diagnostic and infallible and the only method of making sure is by adequate biopsy.

Discharges from the nipple, apart from the physiological secretions of the breast during pregnancy and lactation, are usually either serous or bloody. In either case they are an indication of unusual mammary epithelial activity, but they do not necessarily indicate that a cancer is present. A bloody discharge sometimes occurs in women under the age of 30. It usually originates in an intraduct papilloma which is commonly simple and not malignant and mastectomy in such cases is quite unwarranted. The papilloma itself can usually be detected as a small pea-like nodule, often within two inches of the nipple and pressure upon it will frequently result in a dark blood-like discharge from a single duct opening on the nipple. The duct can then usually be easily entered by a fine lachrimal duct probe, thus facilitating the localization and removal of the papilloma.

In women over 40 a serous or hæmorrhagic discharge from the nipple is not uncommon. It is almost always due to widespread epitheliosis within the breast since it commonly seems to be derived from all four quadrants. Often in such breasts there are several or even many thickenings or lumps, firm but not stony hard, tender and easily felt between finger and thumb. In such a breast, a small early carcinoma can very easily be overlooked and it is therefore difficult to avoid the conclusion that all such breasts should be removed for serial histological study.

In summary therefore the diagnosis of carcinoma of the breast may be obvious on the most perfunctory of examinations or may be impossible to make without biopsy of the suspected portion of the breast. The golden rule should be —make sure; not wait and see.

Clinical assessment.—It is generally agreed that the two most important factors which influence prognosis in patients with carcinoma are: (1) the biological characteristics of the tumour itself, and (2) the degree of advancement or stage which the disease has reached when the patient is first seen.

The biological characteristics of malignant tumours of course differ widely and their accurate assessment is extremely difficult. Cancer of the breast, which is frequently at least partly hormone dependent, is likely to be influenced by age, by pregnancy, by lactation, and by hormonic factors, such as the activity of the pituitary and adrenal glands and the ovaries. In certain cases, it is possible to modify the fundamental growth and metastasizing characteristics of breast tumours by altering their hormonic environment by ovariotomy, suprarenalectomy, or removal of the pituitary gland. These methods are at least of great clinical interest, may be of fundamental importance in the ultimate elucidation of this form of cancer, and, even in their present experimental stage of development sometimes result in great clinical benefit.

The degree of advancement or stage which the disease has reached is of paramount clinical importance in deciding upon which method or methods of treatment will be advised in any given case. It is dependent upon numerous factors, among which are:

1. The biological characteristics of the tumour itself.
2. The known and probable length of history.
3. The size of the primary tumour.
4. The quadrant of the breast in which the tumour originated.
5. Fixation to or actual invasion of the skin by tumour, and,
6. Evidence of distant metastases in bone, in the lungs, in the abdomen, or elsewhere.

Many attempts have been made, based upon these and other criteria to devise a completely satisfactory system of staging in order that the several methods of treatment may be mostly intelligently applied. None is as yet entirely satisfactory, widely applied, or rigidly followed. That this is so is inevitable because any method of staging depends upon evidence which is often quite unreliable, such as the history and our fallible methods of physical examination. How many women assert with complete conviction that the mass in the breast 4 cm. across has only been present for two weeks? How greatly different is the examination of the thin breast and the thin axilla from the massive breast and fat-laden axilla? And how many times must metastases be present in lungs, x-rays of which are described as normal when in fact they are merely within the range of the "commonly seen"?

In spite of these drawbacks, however, clinical staging is absolutely necessary because upon it depends treatment. Its weaknesses and strengths must nevertheless always be remembered both in analyzing and criticizing one's own results and examining the results of others.

Certain data are needed in order that clinical staging may be decided upon. These are:

1. A careful and minute examination of both breasts.
2. Very careful examination of both axilla.
3. Examination of the neck.
4. Careful examination of the abdomen, including rectal and vaginal examinations.
5. Diagnostic chest x-rays.
6. Complete x-ray skeletal survey.
7. Complete physical examination, including such biochemical and other tests as may be thought necessary.

When all this information has been accumulated the clinical stage of the disease can be determined. The following is the classification we prefer:

Stage I.—The tumour is confined to the breast. The skin may be dimpled or the nipple retracted but no invasion of skin is present. No "significant" lymph nodes are palpable in the axilla. No x-ray or other evidence of spread to distant sites.

Stage II.—As in Stage I, but mobile "significant" nodes are palpable in the axilla on the same side only.

Stage III.—The skin is invaded in direct continuity with the tumour and may be ulcerated, or the tumour is attached to underlying muscle and fascia. There may or may not be palpable "significant" axillary lymph nodes on the same side. No x-ray or other evidence of spread to distant sites.

Stage IV.—The growth has extended beyond the breast as shown by: (a) Fixed and matted axillary nodes on the same side. (b) Tumour fixed to chest wall. (c) Secondaries in supraclavicular nodes. (d) Secondaries in the opposite breast or axilla. (e) Distant metastases in skin, bone, lung, liver, etc.

This system of classification of course is far from being all-inclusive. It does however have the virtue of being simple and of being easily applied to the majority of cases of carcinoma of the breast. It also affords a convenient guide to treatment.

Treatment of carcinoma of the breast.—The methods of treatment at present at our disposal are: (1) Surgery—simple or radical mastectomy. (2) Radiotherapy in its various forms. (3) Hormone therapy or hormone withdrawal therapy.

The rôle of surgery.—It is clear, that if in a given case, the tumour is confined to the breast and there is no extension outside it (Stage I) then the removal of the breast by simple mastectomy will cure the patient since it will remove all the disease. It is also plain that in cases in which only adjacent axillary lymph nodes are involved (Stage II) the removal of the breast, the pectoralis major and minor muscles, and the complete clearing of the axilla will also effect a cure, if the disease is not disseminated by the operation.

But, if the disease has reached to the apex of the axilla and beyond it into the posterior triangle of the neck, or if the chain of nodes around the internal mammary artery is involved, or if any other more distant metastasis has occurred, it is also clear that surgical removal of the primary tumour and nearby metastases will not cure the patient of her distant metastases and the operation will have been very largely pointless. Indeed there is evidence, Halliwell[5] (1924) that it may actually disseminate the growth and reduce the patient's survival period.

Thus while it is clear that Stage I carcinoma of the breast is best treated by radical mastectomy, in Stage II, before radical mastectomy is done, it must be shown: (1) that the axilla which contains metastases can be completely dissected without causing further spread of the disease; (2) that Stage II, without spread elsewhere, occurs with reasonable frequency; and (3) that in patients with involved axillary nodes (Stage II) clinical evidence of freedom from distant spread is reasonably reliable.

The first prerequisite was answered by Halliwell[5] in 1924, who found that in advanced cases a better three-year survival rate was obtained by more limited operations than radical mastectomy. He concluded "that an attempt at complete removal which is unsuccessful disseminates the growth and that it is better in very advanced cases not to attempt to clear the axilla".

The second question is, we think, answered by comparing the long-term survival rate following radical mastectomy as the sole method of treatment for Stage II carcinoma of the breast with the survival rate for Stage III cases treated in the same way. Thus Williams, Murley and Curwen[6] of St. Bartholomew's Hospital found that the 10-year survival rate following radical surgery in Stage II carcinoma was 26% and in Stage III it was only 4%. It thus seems likely that radical surgery may save about 20% of cases in Stage II, which would otherwise have been lost, a not inconsiderable salvage rate.

The answer to the third question is perhaps contained in the answer to the second, the much larger 10-year survival rate in Stage II at least

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suggesting that our methods of detecting distant metastases, though fallible, are useful.

Radical mastectomy is therefore indicated in Stage II in at least the early cases of that stage in which wider dissemination is unlikely. But, where axillary involvement is extensive, and especially in cases in which the involved nodes are fixed, it will not only fail to cure the patient but it will also enforce heavier burdens upon her, such as higher operative mortality, œdema of the arm, and more prolonged hospitalization.

In Stage III and Stage IV carcinoma of the breast surgery has very little to offer except occasionally the removal of a fungating tumour which responds poorly to radiation.

Irradiation.—Although radiotherapy for breast cancer was first used by Gocht,[7] as long ago as 1897, it is only in the last 20 years that x-ray therapists have had the important advantages of adequate physical apparatus, sound theoretical knowledge, and sufficient clinical experience. To date few patients with early breast cancer have been treated by irradiation alone,[8, 9, 10] and the results of such treatment have been disappointing, though if given in maximum doses long-term arrest by encapsulation of the disease in scar tissue may occur.[11] It is also possible that x-ray therapy may, when given in inadequate doses, actually disseminate the disease.

The rôle of x-ray therapy as an adjunct to surgery in operable cases, given either pre- or post-operatively, or both before and after operation, still awaits the judgment of long experience. Our present policy in this matter is not to irradiate proven Stage I cases but to give postoperative irradiation to the limit of tissue tolerance to cases in Stage II to the neck and mediastinum.

Radiotherapy plays its greatest part in the treatment of inoperable carcinoma of the breast. Although tedious and expensive in its application, and accompanied by significant morbidities, such as irradiation dermatitis and sickness, it frequently brings much relief from suffering and sometimes very prolonged remission from the disease.

Hormone palliation.—For nearly three-quarters of a century surgeons have been attracted by the possibility of influencing cancer of the breast by oöphorectomy. This concept was advanced by Schinzinger in 1889.[12] The first to apply it was Beatson[13, 14] of Glasgow in the case of a woman aged 33 with advanced carcinoma of the breast. Eight months later all trace of the disease had gone. This case and others enlisted the interest of Alexis Thompson of Edinburgh,[15] Lett[16] and Waring[17] of London and others and, for a time, oöphorectomy occupied a significant place in the treatment of carcinoma of the breast, since it seemed to influence favourably about 20% of cases in which the operation was done.[15]

Irradiation of the ovaries in cases with cancer of the breast was first done by de Courmelles in 1904, who reported his experiences in 1922.[18] Others, including Dresser,[19] Halberstaedter and Hochman,[20] and Raven,[21] using either x-radiation of the ovaries or surgical castration, reported their results and by 1945 it could perhaps be said that the method results in symptomatic improvement in about 50% of cases—bone metastases usually responding well, cutaneous and pulmonary recurrences less favourably, and lymph node deposits barely at all.

The results of oöphorectomy naturally encouraged clinical trials with both androgens and œstrogens by Loeser,[22] Ulrich,[23] Binnie[24] and many other investigators and it is now generally agreed: (1) That œstrogens are of most value for elderly patients with soft tissue metastases. They are only occasionally beneficial to patients with skeletal metastases. In the pre-menopausal patient they often are poorly tolerated and may accelerate the progress of the disease. (2) Androgens are of most value in debilitated patients with osseous metastases. They may in addition be of value in the pre-menopausal woman with soft tissue metastases.

Further and enhanced interest in the biology of tumours was aroused by the coming of cortisone. It made possible surgical removal of the adrenal glands, thereby fundamentally changing the internal environment of the tumour and its host. Suprarenalectomy for malignant disease we owe to the genius of Charles Huggins,[25] who in 1952 reported upon 18 adrenalectomies in patients with late cancer. Among them were seven cases with mammary cancer. One case died in the postoperative period but in the six which lived significant regression in the cancer occurred in three cases.

We have had in the last eighteen months the opportunity to treat five advanced cases of carcinoma of the breast by bilateral suprarenalectomy. In four of the five cases the ovaries were either removed surgically or treated by x-radia-

tion. The essential details of these cases are as follows:

CASE 1

Mrs. J.A.R., aged 39. In 1949 a lump was removed from the left breast. Sections revealed no malignancy. In 1951, a radical mastectomy was carried out for an advanced carcinoma of the left breast. In April, 1952 pain in the right hip. Shown later to be due to secondaries. X-ray therapy elsewhere. Admitted August 20, 1952. Clinically, secondary carcinoma in the right breast with secondaries also in the right humerus, skull, pelvis, right femur. Bilateral suprarenalectomy August 22, 1952. Postoperative course uneventful. ACTH test revealed suprarenalectomy to be complete. Has been maintained since on a dosage of cortisone, varying from 12½ to 25 mgm. per day with desoxycorticosterone acetate implantations of 500 mgm. at about six-monthly intervals. Immediate result of suprarenalectomy excellent. Bone pain disappeared. Radiological evidence of secondaries almost disappeared. Eight months later return of secondary deposits with development of new deposits. Histology of the original tumour spheroidal cell carcinoma of the breast. At present receiving x-ray therapy for secondary deposits in the left femur. Steadily deteriorating. In summary, marked improvement for approximately six months with, subsequently, deterioration.

CASE 2

Miss M.C., aged 40, admitted September 22, 1952 with far advanced carcinoma of the right breast with involvement of skin in the axilla, chest wall, liver, and skeleton. Suprarenalectomy was done on October 1, 1952. Died forty-eight hours later. Original biopsy of breast revealed a scirrhus adrenocarcinoma with spherical cells and deeply basophilic nuclei.

CASE 3

Mrs. S.V.S., aged 55, carcinoma right breast. Classified as Stage II November 1951. Radical mastectomy November 23, 1951. Histology revealed to be a differentiated adenocarcinoma with atypical acini lined by tall columnar cells. Axillary glands not invaded but secondary adenocarcinoma invaded the pectoralis major. Good immediate recovery. Deep x-ray therapy, 4,000r, given through anterior right supraclavicular, posterior axillary, medial and lateral tangential regions. Discharged December 1951. Followed at Clinic. Re-admitted April, 1952, for a further course of deep x-ray therapy to the right supraclavicular, right posterior axillary, tangential medial and lateral right breast. Remained well until August, 1952, when found to have pulmonary metastases and bony metastases on the right side of the pelvis. Further x-ray therapy September, 1952. No improvement. Bilateral suprarenalectomy October 15, 1952, undertaken because of severe bone pain. Suprarenalectomy complete. Maintenance dose of 12½ to 25 mgm. cortisone per day—DCA implantation. Satisfactory control. Very nearly complete recovery from bone pain but condition steadily deteriorated. Patient died November 27, 1952.

In summary, suprarenalectomy did not influence the progress of this lady's carcinoma, but relieved her pain.

CASE 4

Mrs. E.B., aged 32, first admission, under care of another doctor, March, 1948. Tumour of right breast. No malignancy found. Admitted again May, 1950, carcinoma of the right breast, secondary carcinoma in the cervical spine, lumbar spine, and skull. Collapse of the 12th dorsal vertebra. Secondary malignant lesion in the 3rd lumbar vertebra. Bilateral ovariotomy, both ovaries removed. Deep x-ray therapy. Startling improvement—secondaries disappeared. Discharged on testosterone therapy. Readmitted August, 1950. Modified radical mastectomy carried out on August 21, 1950. Biopsy revealed sections of breast to be almost completely replaced by a mass of dense fibrous connective tissue. Skeletal survey in August, 1950, revealed osteolysis occurring in the body of C.4, the 11th dorsal, and the 3rd lumbar vertebra. X-ray therapy given. Remission of pain. General improvement. Readmitted November, 1950—improvement in the secondary metastases. Remained well for twenty-one months, then developed a shower of secondary deposits in numerous bones, associated with bone pain. Bilateral suprarenalectomy August 13, 1953. Discharged August 23, 1953, on testosterone therapy, with grave prognosis. Made a startling recovery and was clinically well for three months. She then became jaundiced and deteriorated very rapidly, finally dying in December, 1953.

In summary, this patient was greatly helped by x-ray therapy and hormone withdrawal therapy.

CASE 5

Mrs. M.N., aged 43, first admission December 12, 1951. Clinical estimate of stage of disease, Stage II. Radical mastectomy December 13, 1951. Biopsy revealed a differentiated adenocarcinoma. All axillary nodes involved. X-ray therapy right posterior axillary, right supraclavicular, and medial and lateral tangential breast. Readmitted July, 1952, for a further course of deep x-ray therapy. Followed closely at the Clinic. Readmitted June, 1953—secondary deposits present in bone. Bilateral oöphorectomy and adrenalectomy in one operation on July 13, 1953. Maintenance dose 12 to 25 mgm. cortisone per day. ACTH Test showed suprarenalectomy to be complete. Readmitted September, 1953—probable secondaries in the 5th rib and left great trochanter. Steady improvement at the present time and now clinically well.

In summary, helped by suprarenalectomy, at least temporarily.

In summary, suprarenalectomy has, we think, done no harm to any of these five patients. It prolonged the life of at least three of them, and where bone secondaries were present produced considerable relief from pain. It is clear that cancer of the breast can be influenced by this measure but perhaps it is also plain that the relief that it affords is short-lived. It is our present intention to carry out ablation of the pituitary gland in some cases of late carcinoma of the breast and look beyond this new horizon, perhaps thereby to learn a little more about cancer of the breast.

SUMMARY

We have at the present time only three weapons with which to treat carcinoma of the breast—surgery, radiation therapy, and hormone palliation. Each has its uses and abuses.

In early cases, surgery may well cure the patient of the disease. In late cases it often adds to her tragic burden.

Radiation therapy rarely harms and is most useful as an aid to surgery and in the "rearguard" actions against advancing disease, sometimes delaying the inevitable for many months or even years.

Hormone therapy in its widest sense changes the environment of the malignant tumour but has at present only a limited usefulness. It may however in the end furnish the key to the disease.

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COLD ACCLIMATIZATION*

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ON EXPOSURE to cold the body has a problem to solve. In its simplest terms the problem is to meet an increased heat loss by increased heat production without alteration, or at least without harmful alteration, of the temperature of the central and vital tissues. Actually, the situation is very complicated. The heat loss may be from the entire body surface or from some localized region. In either event there are certain differentials to be maintained in the blood supply to the tissues underlying the exposed regions. The face, the hands and the feet are the sites of greatest heat loss (in most circumstances) because of their greater circulation, and the circulation to these and related parts requires adjustments of various patterns depending on the detail of the stress to be met.

One facet of the problem is the fact that too great a reduction in the circulation of the extremities so reduces their function that the individual may be incapacitated for many tasks. On the other hand too small a reduction means a needlessly large heat loss. There is, therefore, a balance to be kept between the maintenance of adequate circulation in hands and feet and the necessity of maintaining the temperature of what has come to be called the *core*, the internal tissues whose temperature must be maintained within rather narrow limits if the individual is to survive. This is a problem in the distribution of heat. The production of heat in increased amount is a distinct but related matter, for the manner and degree in which it is produced may depend on the circulatory adjustments which occur. The body has few means of producing more heat to meet environmental demands. It can increase the metabolic activity of visceral tissues or it can increase heat production in muscle by exercise, shivering or simply increase in tone. The size of the contribution by these different means will vary from situation to situation. Finally, the body may, so to speak, release heat from storage, and it is known that on cold exposure a contribution is made to the heat balance of the body by the reduction in temperature sustained by tissues intermediate between the extremities and the core. The tissues involved and the heat they give up depend both on the capacity and opportunity of the body to increase its heat production and on the pattern of its distribution through the vascular tree.

As can be seen, the problem posed to the body on exposure to cold is, even in outline, a delicate and complicated matter.

It is known to some degree how the body reacts to acute cold exposure. For instance, it is a commonplace that there is peripheral vasoconstriction with conservation of heat and that there is increased heat production by shivering and, when possible, by exercise. A fall in the

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level of serum protein-bound iodine which has been noted[1] suggests that there may also be an increase in the metabolic activity of visceral tissues. It is accepted that there is a diuresis and a reduction in both plasma volume and total red cell volume. The release of heat from storage has been reported.[2]

Much less is known about the manner in which the human body reacts to long continued or to repeated cold exposure and most of what is known is of relatively recent discovery. There are still, in fact, those who deny that it reacts at all and who deny that cold acclimatization occurs in man. To those willing to entertain the proposition that cold acclimatization exists in man, a series of questions occur. (1) In the acclimatized is there an alteration of the response to acute cold exposure? (2) In the acclimatized is there an alteration of vascular dynamics which can be observed at comfortable ambient temperatures between acute exposures? (3) If there are changes, by what mechanism are they brought about? These were matters which were investigated by the Queen's University Arctic Expedition 1949 and 1950.[3 to 7]

One of the difficulties facing those who work in this field is the choice of experimental subjects. Until the process is described in physiological terms, those who are cold-acclimatized must be identified in terms of the cold exposure which they have experienced. As it is not yet known how much exposure is required, and as there is evidence that different features of the process appear sequentially as exposure is continued, this means of identification is open to error. It may be remarked here that discussion of the subject has not been helped by the use of the word acclimatization to describe the body response to acute or short term exposure to cold rather than the reservation of it to denote the events which follow long term cold exposure. The selection of experimental subjects from among Eskimos who continue to live in their traditional dwellings and who still gain their livelihood by hunting and trapping has, in the absence of a full physiological description of cold acclimatization, the merit that such Eskimos as these have an ability to live and work in the cold that permits, on the basis of performance, their acceptance as acclimatized individuals.

The Eskimos used by the Queen's group were chosen from among those who live on Southampton Island, Northwest Territories, where the mean daily maximum temperature over a period of 7 years has been in January –16.7°, in February –12.3°, and in March –3.7° F. The average temperature of their tents in daytime during July was found to be approximately 68° F. and in April the temperature in a typical shack was found to range between 17° and 40° F. A control group of unacclimatized subjects was made up of medical students at Queen's University. The Eskimos were studied during June and July and the controls during October and November when the outdoor temperatures in Kingston, Ontario, are approximately those recorded during the summer months on Southampton Island. Only men were used in the vascular studies.

RESPONSE TO ACUTE COLD EXPOSURE

Studies were made of the blood flow in hand and forearm when the limb was immersed in water baths at various temperatures. Blood flow was measured with venous occlusion plethysmographs and tissue temperatures were recorded with needle thermocouples. When a 5° C. water bath was used it was found that the Eskimo maintained at all times a higher blood flow through the hand and forearm than did the controls (Fig. 1). This is probably one reason why they were able to tolerate the water bath for the full 2 hours of the experiment as opposed to the 70 and 90 minutes which marked the limits of tolerance of this temperature by the controls. Examination of Fig. 1 shows that the reduction in blood flow which occurred in the Eskimo was actually greater than in the controls, and that

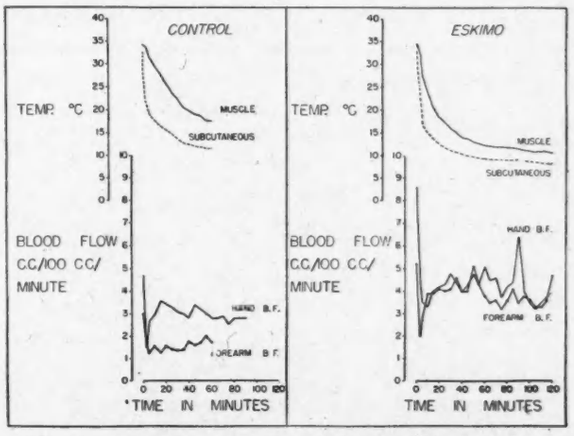

Fig. 1.—Average response of forearm, subcutaneous and muscle temperatures and forearm and hand blood flow in three controls and three Eskimos during immersion of hand and forearm in 50° C. water bath.

the higher level at which blood flow stabilized was the result not of less vasoconstriction in this group but rather the higher initial blood flow. The forearm tissue temperatures, both muscle and subcutaneous, fell more rapidly and to lower levels in the Eskimos, despite the higher blood flow through the forearm. This greater drop in the deep tissues of the exposed limb indicates a greater contribution of heat from storage and is the result of increased cooling of the deep tissues by the greater venous drainage from the hand and the surface of the forearm. During the 70 minute period of the experiments on forearm blood flow the average fall in rectal temperature in the controls was 0.7° C. as compared with 0.5° C. in the Eskimo. At water bath temperatures from 10 to 33° C. the results were qualitatively the same as those at 5° C. and showed a higher hand and forearm blood flow in the Eskimo. In all the experiments spontaneous fluctuations in blood flow were greater in the Eskimos.

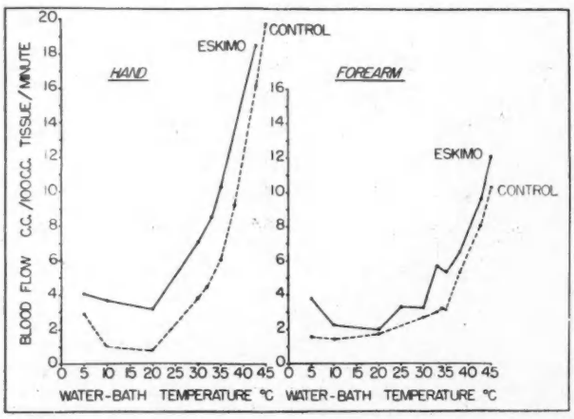

Fig. 2.—Average forearm and hand blood flows in controls and Eskimos in water baths at temperature 5° to 45° C.

These results indicate that on acute local cold exposure there is in the acclimatized a peripheral vasoconstriction which is greater than in the unacclimatized though at all times the blood flow through the hand and forearm, and therefore the heat loss, is greater. More heat is given up by the deep tissues of the forearm by the mechanisms which have been described by Pennes[8] and by Bazett *et al*,[9, 10] In the experiments in the coldest water baths conditions were so severe as to cause a fall in the temperature of the body core, but, despite the greater heat loss from the extremities, the core temperature of the acclimatized was if anything better maintained.

VASCULAR DYNAMICS BETWEEN ACUTE COLD EXPOSURES

Studies of hand and forearm blood flow, skin, subcutaneous, muscle and rectal temperatures were also carried out while the subjects were resting in a room at 20° C. which was a comfortable temperature for the Eskimo. Similarly dressed controls felt slightly cool at this temperature. After a 30 minute period in this room with the hand and forearm clothed with cotton wool, the limb was rapidly bared and skin and tissue temperatures determined. They were all higher in the Eskimo but it was only the forearm skin temperature which was significantly so (Table I).

TABLE I.

TISSUE TEMPERATURE AND HAND AND FOREARM BLOOD FLOW IN ROOM AT 20 C.

	Control	*Eskimo*
Tissue temperatures C.		
Hand skin	32.6 ± 0.89	33.8 ± 0.47
Forearm skin	30.5 ± 0.25	31.2 ± 0.17
Forearm subcutaneous	32.7 ± 0.23	32.9 ± 0.18
Forearm muscle	34.4 ± 0.19	34.6 ± 0.16
Rectal	37.3 ± 0.07	37.1 ± 0.06
Blood flow c.c./100 c.c. tissue/minute		
Hand	4.7 ± 0.19	8.6 ± 0.43
Forearm	3.0 ± 0.08	5.2 ± 0.11

The rectal temperatures were the same in the two groups at the end of 30 minutes but when observations were continued for another two hours the average rectal temperature in the controls fell 0.4° C. while that of the Eskimo remained constant. Observations of hand and forearm blood flows over a 2-hour period showed that in the Eskimo they were significantly higher.

Thus it can be seen that while resting under reasonably comfortable conditions the acclimatized individual maintains a greater blood flow through his extremities than does the unacclimatized. In this situation loss of heat from storage would not be expected, and the deep tissue temperatures are not different. As during acute exposure, the increased heat loss from the extremities is met without prejudice to the core temperatures.

MECHANISM OF THE VASCULAR ADAPTATION

There are similarities between the patterns of peripheral blood flow which have been seen in the Eskimo and those which are seen in hyperthyroidism. Stewart[11] has reported an increased

blood flow through the hand in cases of hyperthyroidism and Eichna and Wilkins[12] found an increased forearm blood flow. Increased spontaneous fluctuations have also been reported.[13] For these reasons B.M.R. determinations have been made on a representative sample of the group of Eskimos from which subjects were obtained for the vascular studies. During July and August, 1949, 48 determinations on 16 subjects gave an average result of +27.4% (DuBois). These results have been described in more detail elsewhere.[3] It may be noted here that Gottschalk and Riggs[14] found an elevation of the serum protein-bound iodine in a group of Eskimos on Southampton Island. It may also be remarked that we have found a marked increase in the circulating blood volume of the Eskimo[7] and that increase in blood volume is also seen in hyperthyroidism.[15, 16]

These facts provide support for the concept that increased activity of the thyroid gland is an essential part of the cold acclimatized state. The now considerable evidence of increased thyroid activity in cold acclimatized animals and man has been reviewed and discussed elsewhere.[7] The present observations on the basal heat production and the peripheral vascular characteristics of the acclimatized suggest that both are related to the heightened thyroid activity.

CONCLUSION

As a result of these experiments it is our conclusion that the vascular response of the acclimatized to acute exposure to cold is different from that of the unacclimatized, that the acclimatized also show changes in the peripheral circulation between acute exposures and that these are in part at least the result of an increased amount of circulating thyroid hormone. In the acclimatized there is an increased metabolic heat production and, perhaps fortuitously, because the increased heat production is due in part at least to increased thyroid secretion, heat is distributed by the vascular system in a manner which maintains function of the extremities at a higher level than would otherwise obtain. It is also the pattern of the vascular changes which accounts for the greater contribution of heat from storage.

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CALCIUM GLUCONATE IN THE INDUCTION OF LABOUR*

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THE COMPLICATIONS of modern living have resulted in increasing pressure on doctors by their patients to elect the date of delivery. Patients who live a considerable distance from the hospital, those whose husbands are members of the armed services and about to be posted outside Canada, those who can engage household help only for a specified and limited period, and many others—all have compelling reasons for requesting their doctor's co-operation in determining the date of delivery. Provided it can be shown that the induction of labour at or near term adds nothing to the maternal and fetal risk, the obstetrician has no good reason to refuse his patient such co-operation, and in our hospital a large number of inductions are recorded as being done for "convenience". The widespread acceptance of this practice as a safe and permissible method of terminating pregnancy has been repeatedly attested in the literature.[1 to 4]

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The time-honoured method of induction by castor oil and quinine has been shown to be ineffective in the case of the former and not without danger in the case of the latter,[5] and there is now sufficient proof that better drugs are available. Among these is calcium, which has been shown to increase the amplitude of uterine contractions, and probably to sensitize it to the action of posterior pituitary extract, either that already present in the body or introduced from without.[6, 7, 8] Our interest in this drug was stimulated by publication of a series of cases by R. M. Grier in 1947[9] and we began its use shortly thereafter. This report concerns 200 consecutive inductions by the method outlined below, all from the author's private practice.

Selection of cases.—In the absence of obstetric or medical reasons for induction, certain requirements must be fulfilled in order to make elective induction both certain and safe: there must be no cephalo-pelvic disproportion, the vertex should present and be engaged, and the cervix must be "ripe", *i.e.*, soft, at least partly effaced, and dilated at least 1 cm. Few primigravidæ fulfill these criteria prior to labour and in general we consider it unwise to attempt elective inductions in first pregnancies. Most of the primigravidæ in the present series were induced for medical reasons.

Method.—On admission to hospital, 10 c.c. of a 10% solution of calcium gluconate* is administered intravenously. This must be given slowly to avoid unpleasant vasomotor symptoms. The injection is repeated in two hours and followed by a large hot tap-water enema. This is immediately succeeded by the intramuscular administration of pitocin m. ii, which is repeated at one-hour intervals for a maximum of six doses or until labour is established. Prior to the administration of each dose of pitocin the nurse is required to make certain by abdominal palpation that uterine contractions are not occurring. If labour is not well established by the 4th or 5th dose it is customary to artificially rupture the membranes.

Results.—In 200 consecutive cases induced by this method there were no failures. Artificial rupture of the membranes was required in only 66 instances, and in almost all such cases labour ensued within two hours. The average number of doses of pitocin was 4.2 or about 8½ minims. There were 36 primigravidæ and 166 multigravidæ in the series and the average duration of labour for the primigravidæ was 8 hours, 35 minutes, and for the multigravidæ 7 hours and 15 minutes. The indications for induction and duration of pregnancy were as follows:

Convenience	166	all more than 38 weeks' pregnant.
Maternal toxæmia	26	34 to 40 weeks' pregnant.
Abruptio placentæ	3	34, 38 and 39 weeks.
Rh isoimmunization	2	40 weeks
Diabetes	1	38 weeks.
Fetal malformation	1	32 weeks.
Maternal brain tumour	1	36 weeks.

There was no maternal mortality. Two cases were morbid due to mastitis. There were four fetal deaths, one anencephalic, one deadborn erythroblastosis fetalis, one premature at 36 weeks induced because of maternal toxæmia and died in 10 hours of atelectasis, and one died late in labour due to prolapsed cord. This baby was full term, weighed 7 lb. 10 oz., artificial rupture of the membranes had *not* been done, and its death does not seem to be attributable to the induction.

SUMMARY AND CONCLUSIONS

Two hundred consecutive successful inductions of labour using calcium gluconate, pitocin, and in one-third of the cases, artificial rupture of the membranes, are reported.

The observations of previous authors concerning the safety and efficacy of calcium as an agent for induction are confirmed.

In those cases which fulfill the criteria for safe induction, the obstetrician can render a valuable service to his patient by electing her date of delivery to suit her convenience.

*The preparation used in most of these cases was Calcium "Sandoz".

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FOREARM FRACTURES IN CHILDHOOD*

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THE COMMONEST SITE of fracture in children is the forearm. Usually such injuries result from a fall on the outstretched arm—a far from unusual event in childhood. Yet our present methods and standards of treatment only too often permit us to make the management of these frequent and simply produced fractures a matter of considerable difficulty and concern.

Case 1 for example presented as a not too unusual or severe fracture. The first post-reduction film showed what was accepted as an adequate, if incomplete, reduction. However, even in plaster this reduction deteriorated so markedly that the surgeon embarked upon an open reduction, only to find opportunity fled and the fracture united. However, during the next three weeks the deterioration continued and the child was eventually released from plaster with a very obvious forearm deformity. No doubt the mother was assured that the bones would gradually straighten themselves. Certainly, the forearm recovered its normal contour within a year but it was not for fully three years that normal body contour by x-ray was established. Unfortunately, for their 3 years of faith and concern the family

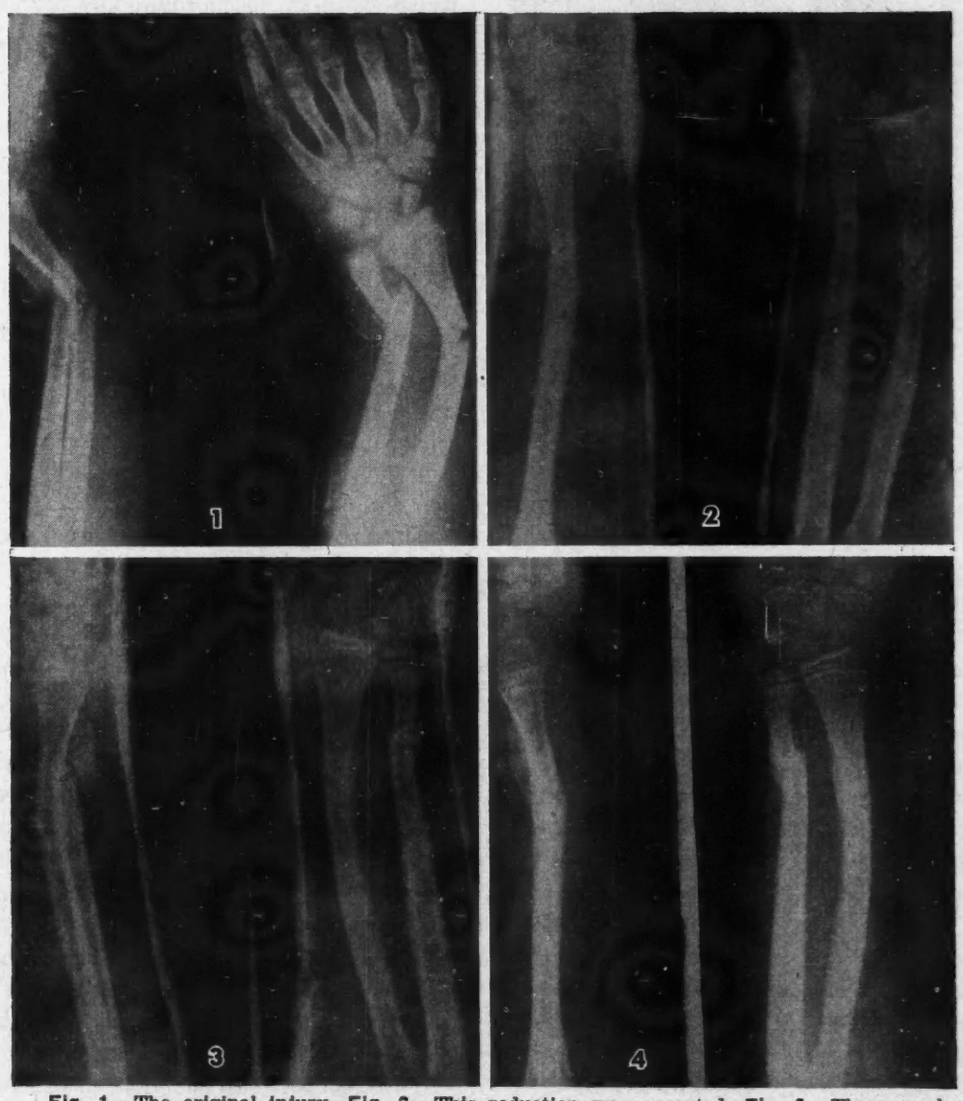

Fig. 1.—The original injury. **Fig. 2.**—This reduction was accepted. **Fig. 3.**—Three weeks later exploration was carried out but the fracture was found to be "united" and accordingly no re-alignment was done. **Fig. 4.**—On removal of the plaster further deterioration was evident.

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Based on a lecture delivered before the Orthopædic Section of the Toronto Academy of Medicine on January 25, 1951.

and surgeon have been rewarded with a child lacking 45 degrees of full pronation.

Quite unsolicited the mother volunteered her opinion that the fracture had never been "set right"—a criticism with which one cannot but concur. Yet by current textbook descriptions of the mechanism of injury and the methods of treatment the surgical management was beyond

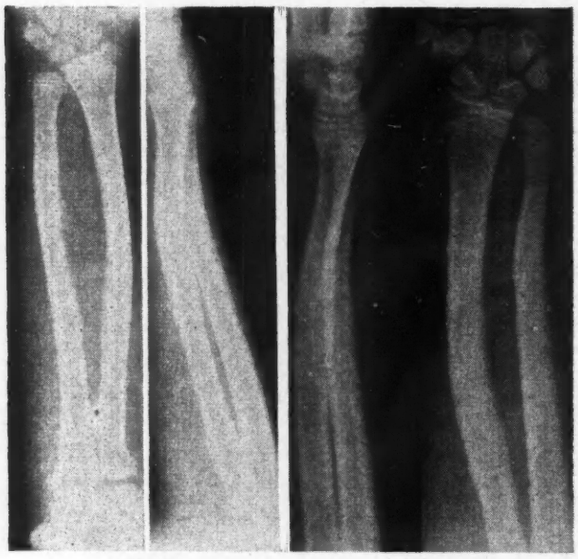

Fig. 5 Fig. 6

Fig. 5.—One year later the arm looks normal and the x-ray appearance is improving. Fig. 6.—Three years later the contour of the forearm is normal, the radiological contour is good and there is a permanent 45° loss of pronation.

reproach. The student will find no clear statement of principles which will prevent him reduplicating the above clinical record. Accordingly I feel our present principles and descriptions of management require re-examination.

In reviewing a considerable number of forearm fractures two very distinct patterns of fracture appeared.

In the first of these patterns, both bones present transverse fractures at the same level. Whether one considers each bone separately or regards both bones and the intervening interosseous membrane as an osseo-ligamentous core of the forearm, we are dealing with a transverse fracture. From our first undergraduate lectures on fractures, we realize that this is a fracture produced by direct violence. In the case of the forearm this force is usually a direct blow (the forearm being struck by a moving object; or the forearm being swung against a solid object) and less commonly a fall on the hand in which the forearm is swung directly against the ground much as an axe is swung into a tree trunk. There are two important points about this type of fracture—firstly it is easily reduced by reversing the apparent radiological deformity, easily controlled and rarely gives rise to any concern. Secondly it is not a very common type of forearm injury. Most textbooks by inference, if not direct statement, nominate direct-violence-transverse injuries as the usual type, a suggestion which is as misleading as it is inaccurate.

A second pattern of injury—due to rotational forces—is much more commonly found. Fig. 7 is

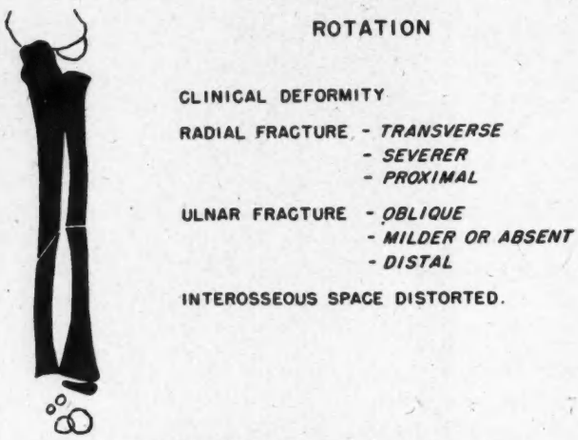

Fig. 7.—The features of a "rotation" fracture.

a diagram of this second pattern as it is found in the midforearm. In the antero-posterior x-ray we observe several distinctive features not all of which, of course, will be invariably present. While both bones are fractured, the fractures are at different levels and are of different types. The radius is usually the more severely fractured and it is fractured transversely. The ulna, however, is usually fractured obliquely and less severely. While we could explain the transverse fracture of the radius in terms of direct violence, we recognize (again from fundamental principles) that the spiral fracture of the ulna can result only from a twisting injury. If we consider both bones and interosseous membrane as the core of the forearm, the fracture lines together run an oblique course across this core, again requiring a twisting force as the only possible explanation of the fracturing mechanism.

The interosseous space is subject to individual variation, but it is always a single compartment bounded by long smooth curves. Any degeneration of this space into an hourglass configuration bounded by one or two sharp double curves bespeaks gross abnormality. Admittedly such a deformity could be produced by lateral angulation, yet usually such angulation is not demon-

strable and there remains solely the conclusion that we are viewing simultaneously two aspects of the same bone—a condition which could be produced only by rotation.

If we are to postulate that these fractures are produced by a rotational force we must explain how a fall on the outstretched forearm can produce this specific force. The explanation lies in a reversal in the usual balance of action. The child is moving forward as the fall commences.

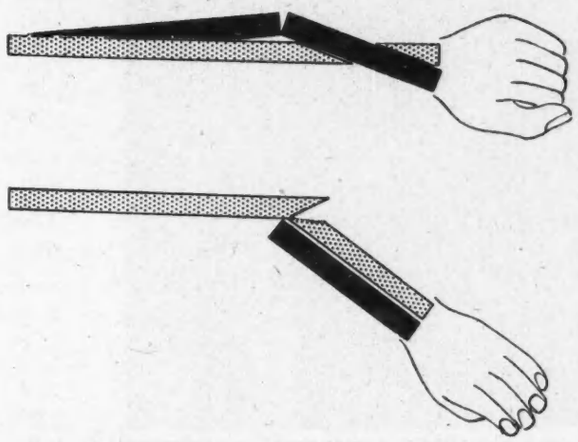

Fig. 8.—Fracture by pronation—a diagram of the mechanisms involved.

When the hand strikes the ground it is firmly fixed by the momentum of the body. Not only does this momentum fix the hand, it carries the body forward and around the locked hand, thereby producing rotation of the hand relative to the body. If you place your hand firmly on top of a desk and holding it fixed walk forward past the hand there will remain no doubt as to whether such a manœuvre does in actual fact produce forearm rotation.

Continuing what has so far been a purely theoretical discussion, how can a rotation force produce a transverse fracture of the radius? This occurs at the extreme of normal rotation when the radius on being forced beyond its normal limits is fractured across the ulna much as one cracks a piece of kindling across one's knee. Fig. 8 represents diagrammatically such a mechanism in the case of a pronation injury. In pronation injuries it is easy to visualize the radius being cracked across the ulna. In supination injuries where the radius does not actually cross the ulna, the modifying influence of the interosseous membrane which is tightness in supination presumably produces the same effect as if the radius actually had rolled across the ulna. Should rotation continue the ulna as the sole stabilizing influence bears the entire brunt of the rotational injury and accordingly is fractured obliquely.

Is there any evidence to support this description of sequence of events? Direct experimental evidence is lacking as this childhood group obviously presents an extremely limited field for autopsy-room study. There are, however, several observations which would confirm such a pattern:

Firstly, isolated fractures of the radius are not uncommon while isolated fractures of the ulnar

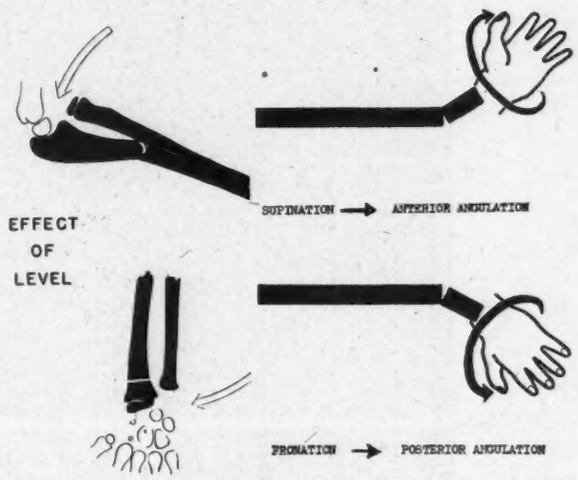

Fig. 9 Fig. 10

shaft are rare. This would suggest that the radius is commonly exposed to the major trauma. Further, in the majority of instances in which both bones are fractured, the ulnar fracture is the milder. A very common pattern is a complete fracture of the radius with a greenstick fracture of the ulna. If both bones are completely fractured it is usually the radius in which apposition is lost. A complete fracture of the ulna with a greenstick fracture of the radius I have seen only at the wrist where the ulnar fracture is of either the styloid or epiphysis.

Before leaving the antero-posterior film another consideration—the effect of level—merits attention (Fig. 9). The two bones are injured at different levels. In the midforearm the radius is usually fractured at a higher level than the ulna. This relationship is the usual arrangement throughout the forearm. However, towards the extremities of the segment the injury may appear an isolated fracture of one bone, the injury to the other bone being dissipated in the terminal ligaments. The injury to the ulnar collateral ligament of the wrist in apparently isolated distal radius fractures, rarely becomes of major clinical moment. What is important is that

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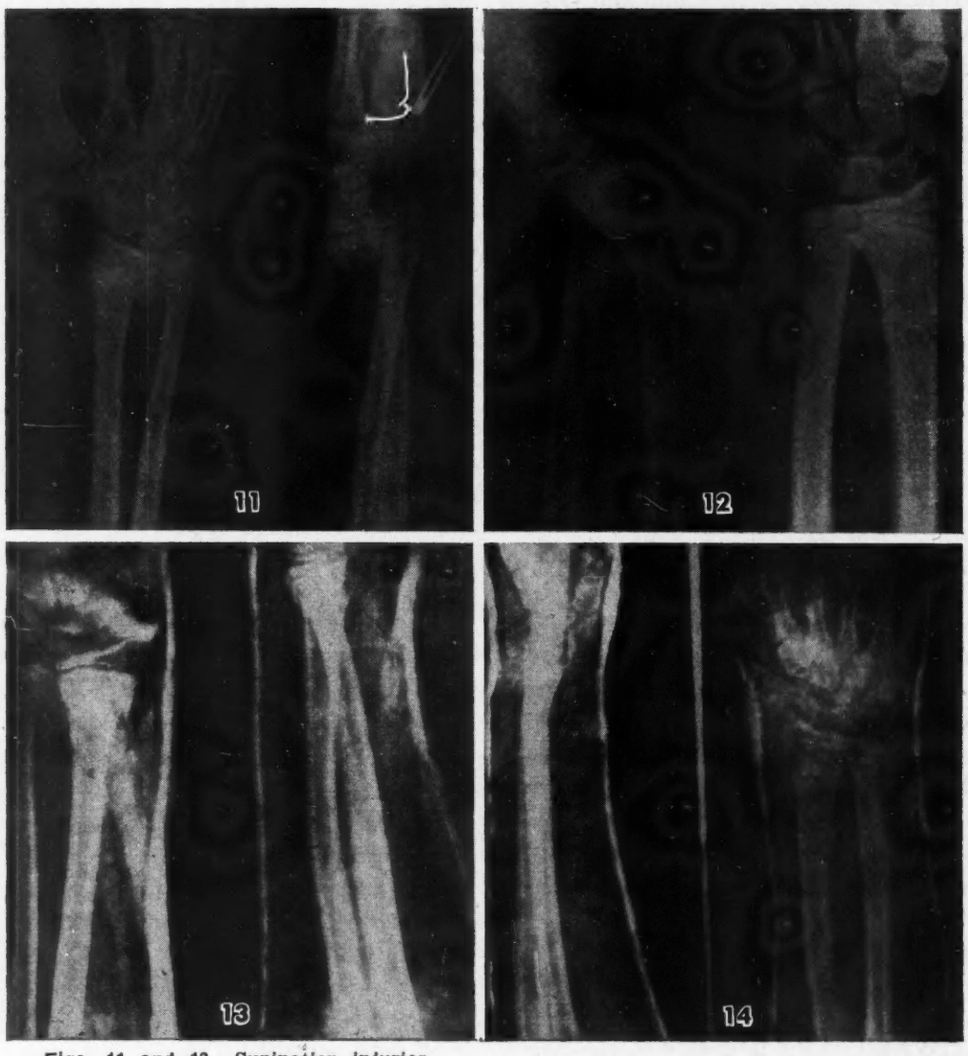
a similar injury at the other end of the segment be recognized for what it is—a Monteggia fracture rather than an isolated ulnar shaft fracture. Of course single bone fractures can occur provided the fracturing ceases at the appropriate moment, yet this possibility does not exempt us from deliberately ruling out the second injury.

the steps leading to the development of the deformity. In which direction would you wind your own forearm to break the radius across the ulna and produce the deformity you see on x-ray? As a rule of thumb confine your interest to the radius and visualize the radius as actually being broken across the ulna. Pronation injuries will be charac-



Figs. 11 and 13.—Supination injuries . . . . .
Figs. 12 and 14.— . . . . . and pronation injuries can occur at the same level. They are immobilized in opposite positions.

So far we have been able by consideration of the antero-posterior view alone, to establish the diagnosis of rotational fracture. There remain two factors which will enable us to establish the precise nature of the rotational force, pronation or supination. Occasionally the forearm will present a rotational deformity, the hand lying in obvious pronation or supination. This however, is not always present or marked. The lateral x-ray, however, will invariably give the key. To interpret this view, retrace in your mind's eye

terized by posterior angulation. In supination injuries the angulation will be anterior (Fig. 10). There is no point in memorizing these relationships; it is perfectly simple to carry out a phantom manipulation on your own forearm. Keep in mind that the radius is to be broken across the ulna.

Once the diagnosis of rotation injury and the specific type is made the principal manœuvre in reduction is obvious. Major correction will be achieved by reversing the fracturing force—

pronation fractures should be reduced principally by supination and vice versa. Flexing the elbow to a right angle and placing one hand firmly around the forearm above the fracture while the other hand grasps and rotates the hand and wrist will permit adequate control for reduction of undisplaced fractures. To demonstrate the specificity of the manœuvre we ran a small experimental series using the one manœuvre only with excellent anatomical reductions. However, there is every reason to include judicious traction and angulation in the reduction manœuvre provided it is recalled that the principal manipulation is one of rotation. These fractures are not pulled out or pushed back—they are wound on.

The more severe fractures and displacements cannot be so easily controlled. Over-riding of the radius may be very difficult to reduce by manipulation although it can usually be replaced by increasing the original deformity and engaging the cortices by a combination of distal traction and direct local pressure before reversing the rotational element and locking the fragments home (such a fracture is demonstrated in Figs. 11 and 13). Should this fail open reduction should be carried out. Once the fracture is reduced it can usually be demonstrated as stable in the appropriate type of rotation—accordingly internal fixation is not often necessary.

Once reduced these fractures should be held immobilized in the appropriate type of rotation. Adequate control of rotation can be maintained only by plasters extending from the palm of the hand to well above the elbow. While there may conceivably be room for argument about the necessity for immobilizing very low radial fractures in above elbow splints (and the chubbiness of a child's forearm is a very potent argument for above elbow plasters in fractures at any level), there is no possible argument about the necessity for immobilizing the elbow in all double-bone fractures and all radial fractures distant from the wrist.

It is important to realize that there is a distinction to be made between the greenstick fractures of children and the more complete fractures of adults. In the adult the relative paucity of corrective molding makes anatomical reduction even more imperative, yet the adult fractures do not respond so accurately to manipulative reduction—control of an adult fracture is considerably more difficult and complex a problem.

SUMMARY

1. Rotational fractures are the common type of forearm fractures.
2. The features of such fractures are fairly clear cut.
3. The nature of the rotational force can be estimated by a phantom manipulation on oneself.
4. Manipulation using principally rotation in the opposite direction will, in the great majority of cases, produce accurate reduction.

The earlier phases of this study were carried out at the Royal South Hants and Southampton Hospital in collaboration with Mr. Ross Bloom to whom I am more than ordinarily indebted. Further work was conducted at the Massachusetts General Hospital where counsel and stimulus of Dr. E. F. Cave and his colleagues in the Fracture Unit provided inestimable encouragement.

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DIET AND ACUTE LIVER DAMAGE*

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THE DETERMINING INFLUENCE of diet for good or ill health is supported by many examples in both human and animal nutrition. Of the numerous incapacitating lesions that may readily be produced experimentally by malnutrition—loss of sight, sterility, nephritis, kidney and urinary calculi, adrenal cortical necrosis, gastric ulcers, muscular dystrophy, myocardial degeneration, atherosclerosis, anæmia of various types, lesions of nerve and skin tissues, caries, skeletal deformities, "congenital" (fetal) abnormalities, fatty liver, and liver cirrhosis—all leading to impaired growth or loss in weight and to premature death, none is more dramatic and more quickly lethal than the lesion termed acute massive liver necrosis. Since it was only a few years ago that Sellers[1] published an excellent paper in this

*Contribution to the Queen's Medical Centenary Number of the *Canadian Medical Association Journal* from the Department of Biochemistry, Faculty of Medicine, Queen's University.

Journal in which the broad field of dietary factors and hepatic injury was briefly reviewed, the author will restrict the scope of the present paper to a consideration of acute massive liver necrosis and will only refer to other aspects of diet and liver damage in order to orient the reader in the field.

Although in retrospect, as was subsequently pointed out by Himsworth and Glynn,[6] there appears to be little doubt that the first investigator to stumble upon the lesion of acute massive liver necrosis in rats was Weichselbaum[2] in 1935, it is also clear from his paper that he was not aware of the exact nature of the lesion and there is no indication that he attempted to ascertain it by histological study. Post mortem examination of the experimental animals revealed "definite hæmorrhages throughout the liver". The inclusion of 0.2% cystine or an iso-equivalent amount of sulphur in the form of methionine prevented the death of the rats. The significant feature of this work, which was not realized until some years later, was the fact that the lethal liver damage had been induced by feeding a diet low in protein and specifically low in the sulphur-containing amino acids, methionine and cystine.

A few years later, Gyorgy and Goldblatt[3] noted that in a number of rats maintained on a diet deficient in the vitamin B complex but supplemented with thiamin, riboflavin and pyridoxine, a few died of acute liver damage. Others showed merely some degree of fatty infiltration and fibrosis. They described the livers in which the acute abnormality was present as showing a yellow, red and brown mottled appearance. Histological examination revealed the presence of necrotic areas varying in size from relatively small areas of necrosis situated primarily around the central veins to confluent lesions involving many contiguous lobules and in some cases extending throughout almost an entire lobe. They assumed that the cause of the lesion was nutritional in origin and quite logically suggested that a deficiency of some component of the vitamin B complex was probably responsible. It is of importance to note that in this initial work the basal diet was not low in protein (casein 18%). In a later investigation the casein level was decreased to 10% and the authors[4] found that the lesions of liver necrosis and cirrhosis then occurred regularly rather than sporadically as had been the case in previous work. They concluded that the pathogenesis of liver necrosis and cirrhosis was related to the lipotropic action of casein and in 1942[5] suggested that the necrotic stage observed by them was an intermediate phase in the development of liver cirrhosis.

The first real advance towards an understanding of the etiology of acute massive liver necrosis was made by Daft, Sebrell, and Lillie[9] in 1942 when they noted that whereas the lipotropes, choline, and methionine—a compound that enables the body to synthesize choline—prevented fatty infiltration and cirrhosis, cystine potentiated these lesions; methionine or cystine prevented the development of necrosis and choline had no influence on this abnormality. On the basis of these findings they postulated that the necrosis and cirrhosis described by Gyorgy and Goldblatt,[3, 4, 5] were separate and distinct entities. This hypothesis was fully substantiated first of all by Himsworth and Glynn[6] in 1944 and subsequently by a number of other workers. In their investigations Himsworth and Glynn utilized yeast as a source of dietary protein and noted that it produced a higher incidence of necrosis than did diets containing equivalent amounts of protein in the form of casein. It should be noted here that, unknown to the English workers, Hock and Fink[12] had previously (1943) discovered that acute liver necrosis could be produced in rats on certain diets in which the protein was supplied in the form of various types of yeast. They found that the necrogenicity of the yeast corresponded fairly closely to the nitrogen-sulphur ratios, those yeasts having the larger proportion of sulphur being much less necrogenic. Himsworth and Glynn discarded the possibility that the yeast contained a toxic substance on the basis that increasing the level of yeast in the diet was effective in preventing the development of the liver lesion a fact also noted in our laboratory.[7] That this should be so is not surprising since Gyorgy and Goldblatt[3] in their original paper pointed out that the addition of yeast or yeast extract always prevented the hepatic injury. A few years later, Gyorgy, Rose, Tomarelli and Goldblatt[8] were unable to explain the differing necrogenic properties of various types of yeast on the basis of their content of the sulphur-containing amino acids, or of any other substance known to influence the process at that time, an experience essentially similar to that of Lindan and Work,[10] and they suggested that the differing necrogenicities of the various yeasts

might be due to the presence of varying amounts of a toxic factor. However, certain work performed by McLean and the writer indicates that although there is probably an element of truth in both points of view, neither one is completely adequate. There would appear to be no gainsaying the fact that there is present in yeast a protective factor, otherwise increasing the level of yeast in experimental diets would not prevent the appearance of acute liver damage.

The suggestion that yeast contains a toxic factor as put forward and supported by Gyorgy *et al.*,[8, 27] rests on much more insecure grounds and might well be disregarded entirely were it not for the following evidence obtained by us. In one experiment two groups of rats were fed a basal diet containing 18% yeast but in one diet the yeast was extracted with hot alcohol prior to incorporation in the diet. This alcohol extraction removed approximately 1.8% of lipid. The incidence and times at which necrosis occurred were for the extracted and non-extracted yeast diets respectively: 1/10 at 50 days, and 7/10 at an average of 59 days. These figures would appear to indicate beyond question that some toxic factor—and we suspect a component of the lipid fraction—had been extracted from the yeast. Apart from the fact that extraction with hot alcohol would extract lipids and compounds of similar solubilities, the belief that the necrogenic material may be a member of the lipid family of compounds is based primarily on our finding[7, 11] that increasing the dietary levels of lard potentiates the necrogenic effect of experimental diets. A further piece of information in support of this belief is the fact that extracted and non-extracted yeast when incorporated into a diet along with the same amount of fat have the same necrogenicity whereas, as we have just noted, if they are incorporated into a diet without added fat the non-extracted yeast diet (containing yeast lipid) leads to a relatively high incidence of liver necrosis and the extracted yeast diet (free from yeast lipid) has a much lower necrogenic effect. There would therefore appear to be fairly good grounds for the belief that yeast contains one or more types of protective substances and in addition a necrogenic factor associated with the lipid fraction.

This circumstance might serve to explain why a higher incidence of liver necrosis has been consistently observed when diets containing yeast as a source of protein were utilized in place of an equivalent or smaller amount of protein supplied in the form of casein. In one experiment[7] performed in our laboratory it was noted that although an 18% yeast diet supplied about 7.2% protein and provided about twice as many milli-moles of amino acid sulphur as did a ration containing 5% casein, all of the animals receiving the yeast diet died of acute liver necrosis whereas none of those on the casein ration developed acute liver damage and all survived the experimental period of 172 days. Another possible explanation of this phenomenon is to attribute prophylactic properties to some other amino acid in casein or to a substance associated as an impurity with this protein. The latter possibility is at present being investigated by Schwarz, recently of Germany, at the U.S. National Institutes of Health, Bethesda, Maryland. Schwarz,[13, 14] in 1944, had discovered that the incorporation of casein, pretreated with alkali, into certain diets caused the production of acute liver damage, a finding that was not picked up by other workers in the field until two or three years later due to the disruption of the usual channels of communication by the war. This lesion, although apparently similar to the lesion first described by Gyorgy and Goldblatt,[3] was found by Schwarz[13, 14] to be not affected by the sulphur-containing amino acids and to be prevented by xanthine and by alpha-tocopherol. The last feature was to assume significance in the light of subsequent developments. The first feature alone serves to indicate that the etiology of this type of liver necrosis is different to that being discussed in this paper, a conclusion with which Schwarz[16] himself also concurred. As a further indication, Abell and Beveridge[15] showed that xanthine was without effect in preventing the hepatic necrosis that develops in rats fed rations low in methionine and cystine. Since Schwarz' arrival on this continent about five years ago he has been engaged in an intensive study of the acute liver damage that develops on diets deficient in the sulphur-containing amino acids and in alpha-tocopherol. Part of this work has been concerned with an attempted isolation of a protective factor from casein, which he has termed factor 3. Potent extracts[17, 67] have been prepared but the identity of the active material remains to be determined.

Following the earlier reports by Gyorgy and Goldblatt,[3, 4, 5] Daft, Sebrell, and Lillie,[9] and Himsworth *et al.*,[6, 18, 19] a number of competent

investigators[20 to 24] attempted to produce liver necrosis by using diets similarly deficient in protein and in the sulphur-containing amino acids but failed to obtain comparable results. Indeed the efforts of most investigators during this period were marked by a high degree of inconsistency and even led to speculation that some infective agent might be primarily responsible for the condition. This possibility had been considered and investigated by Gyorgy and Goldblatt[3] in their original paper and later by Himsworth and Glynn.[6] These workers were unable to implicate any infectious factor in the production of the lesion. An indication of the state of affairs at this time was given by Gyorgy and Goldblatt[25] who in discussing a decade of study on dietary hepatic injury, stated: ". . . we were unable to produce necrosis at will and in this respect have not made any substantial progress beyond our first observations".

This inconsistency in results led us, in 1948, to make a critical comparison of the diets used by the different groups and it was noted that all rations producing a relatively high incidence of hepatic necrosis contained cod liver oil as a source of vitamins A and D; the fat component was low in alpha-tocopherol and furthermore this substance was not included in the vitamin supplements. These points were taken into consideration and it was found that the lesion could be readily and consistently produced and with an incidence higher than any previously published (100%).[26]

The fact that alpha-tocopherol was effective in preventing the hepatic lesion was unequivocally established by Gyorgy[28] whose attention was drawn to this possibility by the results of some of his early experiments in which the incidence of liver necrosis was found to be much less when Crisco, a vegetable fat rich in vitamin E, was substituted for lard. This finding was made independently to that of Schwarz[13, 14] whose report on liver damage due to alkali-treated casein was not available to workers outside Europe until about 1946. Further confirmation of the prophylactic rôle of vitamin E was made by Himsworth and Lindan,[29] Gyorgy and Goldblatt,[30] and Abell and Beveridge.[31] Although this comprised a distinct advance in the field and afforded an explanation for some of the inconsistent results obtained by various investigators nevertheless it appears obvious that still other factors play a potent modifying and perhaps determining rôle in the production and prevention of acute liver necrosis due to dietary causes. Over the past few years a number of these modifying influences have been discovered and most of them are discussed below.

The initial weight of the rat when placed on the experimental ration has a marked effect on the time at which the acute hepatic damage occurs. In one of our experiments[15] groups of rats of average initial weights 53, 129, and 235 gm. died of liver necrosis at the following average times: 38, 58, and 205 days respectively. This result indicates that normal rats have a reservoir of one or more substances that prevents the development of liver damage under these conditions and/or that the demand for the protective factors (methionine, cystine, alpha-tocopherol, etc.) decreases with increasing age. The influence of sex was also determined and it was noted that although the incidence of necrosis was essentially the same in both sexes the average time at which death occurred due to liver damage was significantly longer in the females (79 days for the females vs. 58 days for the males, $P = < 0.01$). The difference in the response of the two sexes obviously may reside in their divergent hormonal environment; or differences may exist in the protein requirements of the two sexes, a possibility for which evidence was adduced previously by the writer.[32] It is of interest to point out that Weichselbaum[2] in his original paper commented on the fact that the mortality was definitely less in the female rats. Both Gyorgy[33] and Schwarz[34] have reported similar findings.

The latter investigator also showed that cortisone ameliorated the necrogenic effect of the basal diet. The only other hormonal secretion that has been shown so far to play a rôle in the process under review is that of the thyroid gland. Handler and Follis[35] first demonstrated that desiccated thyroid greatly accelerated the appearance of acute liver damage, a fact also noted and extended by us.[15, 36] Both propylthiouracil and potassium thiocyanate, representatives of the two large classes of anti-thyroid agents as designated by Astwood,[37] had no effect on the incidence of acute hepatic damage but did significantly prolong the length of time required for death to occur due to this lesion.[36] The addition of cystine or alpha-tocopherol to the basal diet containing desiccated thyroid did not overcome the well-known thyrotoxic effects due to

excessive amounts of this tissue but the rats lived longer—indeed three rats survived the experimental period of 105 days—and none of the animals in these groups developed acute liver injury. The fact that both alpha-tocopherol and cystine prevented liver necrosis in hyperthyroid rats led us to conclude that a high metabolic rate *per se* is not a prime factor in the pathogenesis and that the level of thyroid activity is a subsidiary not a determining factor in the production or prevention of this hepatic lesion. We were unable to implicate the pancreas or spleen one way or another in the process under study.[38]

In two sets of experiments[15, 26] we demonstrated clearly that the inclusion of only 2% cod liver oil in the basal diet significantly increased its necrogenicity. Thus when amounts of crystalline vitamins A (acetate) and D (calciferol), equivalent to the amounts of those substances in 2% cod liver oil were used in place of the latter, the fat level being maintained constant by an appropriate increase in the lard moiety, the incidence of hepatic necrosis decreased from 95 to 55% and the length of time required to cause death due to liver damage increased from 58 to 74 days ($P = < 0.01$). It is probable that the effect of cod liver oil is due to its antagonistic effect on the action of alpha-tocopherol, a phenomenon described by Mackenzie *et al.*[39]

We have made fairly extensive alterations[7, 15, 26] in the salt and vitamin mixtures without implicating any of their components with the exception of vitamin K which significantly prolonged the length of time required for the lesion to develop. Perhaps special reference should be made to the fact that neither Gyorgy and Rose[40] nor ourselves[15] could demonstrate a definite effect from supplementing the basal ration with vitamin B_{12}. In the latter investigation both parenteral and oral routes were utilized.

In an attempt to elucidate further the etiology of the lesion and in order to facilitate subsequent experimental work McLean and Beveridge[7, 42] carried out a systematic investigation into the effects of varying the dietary levels of fat, protein, and carbohydrate. The protein component was varied by feeding yeast at levels of from 0 to 60% and casein at levels of from 0 to 7%. No acute liver damage occurred in groups fed rations devoid of protein or containing casein at levels of 1 to 2%. At concentrations of 3 and 4% casein there was a low incidence of hepatic necrosis and none at levels greater than 4%. At that time we suggested that:

> ". . . at low levels of protein the general level of metabolic activity is so low that an insufficient amount of the toxin responsible for the acute liver injury is produced, or a sufficient amount of the substances responsible for the detoxification process is available or becomes available due to catabolism of body tissue. At higher levels of protein, the general level of metabolic activity is raised and, due to the increased demand for protective factors, such as methionine, cystine and alpha-tocopherol, relatively less of these substances are available to preserve the liver in a normal state, hence fatal liver damage occurs. With a further increase in protein, some of these protective factors are present at a sufficiently high level to overcome the effects of the toxin responsible for the hepatic necrosis".

It may be noted that this explanation is analogous to that first put forward by Griffith and Mulford[41] in connection with their study of conditions affecting the accumulation of fat in the liver due to a dietary deficiency of lipotropic factors. In the yeast series at concentrations of 30% or above, no liver damage was observed. Diets containing between 5 and 18% yeast caused the death of all animals due to liver necrosis. In the fat series, the necrogenicity of the diet was progressively increased by increasing the fat content. Furthermore a diet free from fat in which the protein was supplied in the form of alcohol-extracted yeast did not lead to acute hepatic injury despite the fact that the experiment was carried on for a period of 172 days and the rats on a diet containing 5% lard, but otherwise similar, died at an average time of 73 days. In the same year (1951) Dam and Granados[43] arrived at the same conclusion following an experiment that lasted only 70 days. Since it seemed apparent that some constituent of the fat moiety of the diet either potentiated or was necessary for the production of liver necrosis, fractionation studies were performed on the lard in an attempt to isolate the necrogenic material. During subsequent feeding experiments[44] carried out in succession over a period of 15 months the fat-free ration was utilized several times and, much to our surprise, led initially to a low incidence of liver necrosis which tended to increase with each dietary trial, the incidence being: 1/10, 3/10, 8/9, and 4/10. The possibility that seasonal variations might have been responsible for these results has been considered and discarded on the basis that the experiments covered a period of about 15 months and for the additional reason that during many previous experiments carried out in this field during six years no effect of season has ever been observed on the develop-

ment of liver necrosis. Gyorgy, Stokes and Goldblatt[45] were also unable to show that seasonal variation played a rôle in the production of this lesion.

Although Naftalin[46] has shown that of the three ranges of environmental temperature studied, 60 to 64° F., 70 to 78° F., and 88 to 92° F., the best temperature for the production of acute liver necrosis was 70 to 78° F., this work has little significance in the present instance since the animal room was maintained at a temperature of 68 to 72° F. excepting during the summer months when the temperature usually ranged between 68 to 80° F. The possibilities that changing dietary ingredients and/or changing susceptibility of the animals have been also considered and discounted as explanations for the results due to the fact that there was no progressive increase in susceptibility to the development of hepatic damage in the animals receiving diets containing fat. In casting about for a rational explanation of our results we were impressed by the striking similarity between our experience and that of Gyorgy *et al.*[45, 47, 48] in their work on the ameliorative effect of certain antibiotics (*e.g.* aureomycin) on the development of dietary hepatic necrosis. They found that when aureomycin was used in successive experiments it gradually lost its anti-necrogenic property—a situation that is somewhat analogous to our own experience in utilizing diets free from fat at successive intervals.

In another paper this group[48] suggested that the anti-necrogenic action of aureomycin is due to its

> ". . . suppression of the intestinal flora, or at least of some of its constituents, and thus prevents the formation of bacterial metabolites with which the liver, in the absence of vitamin E or of sulphur-containing amino acids as detoxifying agents, is unable to cope".

The progressive decrease in the delaying effect of aureomycin noted in successive experiments led them to postulate

> ". . . that in the course of 1 and ½ years either aureomycin resistant strains of intestinal bacteria developed in the animal room and were transmitted from one experiment to the following one or that the virulence of some unrecognized infectious factor, such as a virus, increased to such an extent that the aureomycin effect was obscured."[45]

The former sequence of events might well be responsible for the results obtained with the feeding of the diet free from fat to successive groups of rats. Regardless of the validity of the foregoing hypothesis, there can be no doubt that the intestinal flora *directly or indirectly* plays an important rôle in determining the susceptibility of rats to the development of dietary liver necrosis. This statement is firmly supported by Gyorgy and his colleagues[45] on the effect of various antibiotics and on germ-free experiments carried out in collaboration with Reyniers and his associates in the Germ-free Life Laboratory at the University of Notre Dame. The germ-free rats lived twice as long as the control rats outside the germ-free laboratory and at autopsy no necrosis of the liver was found. Work along these lines is still in progress in that institution. Further weight is given to the hypothesis that the intestinal flora may be involved directly or indirectly in the production of the lesion by the fact that the left half of the liver is usually more severely involved than the right half[6, 49] and, as has been demonstrated by Copher and Dick[50] and Glynn and Himsworth,[51] the portal blood from the left half of the large intestine, a segment with a relatively high bacterial count, drains into the left half of the liver. The latter workers, however, believe that the predilection for the lesion to be more severe in the left half of the liver may be explained by the striation of portal blood flow causing most of the protective nutrients absorbed from the small intestine to be channelled into the right half of the liver.

The wide dissimilarity in molecular structure of alpha-tocopherol and the sulphur-containing amino acids, methionine and cystine, and the fact that these compounds exert a similar prophylactic action in the prevention of acute liver necrosis has led to much speculation on the relationship between them and on their mode of action. One explanation is that these compounds may be required at different points in a particular metabolic process. The possibility also exists that one of these substances might act exclusively within the intestinal tract and another in some part of the body after absorption. McLean and the writer[52, 53] administered these compounds by mouth and by injection in the hope that this experiment might throw some light on the site of action and on the intestinal toxin theory to which reference has just been made. Since all three anti-necrogenic agents had essentially the same effect by both routes of administration no definitive information was gained on the sites of action of the individual substances but the results indicated that they probably did not act exclusively in the gastro-intestinal tract. The fact

that certain compounds when administered parenterally can enter the lumen of the digestive tract must of course be kept in mind. Since the insolubility of cystine precluded its administration by injection, cysteine, generally accepted as nutritionally equivalent, was used. The prophylactic effect of cystine was surprisingly low inasmuch as an amount equivalent to 0.5% cystine although prolonging the length of time required for the development of liver necrosis (61 vs. 39 days), did not prevent the lesion which appeared in all animals getting cysteine by mouth. It was also noted that cysteine had a much less marked effect than methionine on a mole: mole basis.

After this experiment was completed, Schwarz,[54] in a paper delivered at the 1952 meeting of the Federated Societies, also commented in a similar vein on the relative effects of dietary cysteine and cystine. When one considers that the anti-necrogenic effect of methionine is thought to be due to its conversion to cystine in the body it becomes difficult to explain the relatively poor anti-necrogenic action of cysteine compared to methionine since the latter amino acid, in order to give rise to cystine, must presumably first of all form cysteine.[55] Our result raises the interesting possibility that methionine may not need to be converted into cystine in order to exert its prophylactic action. Since both Schwarz and ourselves find that cysteine is much less effective in preventing liver necrosis than is cystine one is led to the conclusion that under the dietary conditions used the conversion of cysteine to cystine is impaired.

Workers in this field are amazed at the rapidity of the change from a state of apparently fairly good health to a moribund state within a matter of a few hours of the onset of the lesion. The first manifestations of illness are a listless dumpy appearance, ruffling of the fur and a yellowish, orange stain around the urethral meatus. The condition of the rat deteriorates rapidly, body temperature falls, and death usually occurs within one day of the first appearance of the illness although some animals may last for two or three days. Various histological and chemical studies have been carried out in attempts to discover some early change or sign which would enable one to predict that liver necrosis would supervene unless the dietary conditions were appropriately altered. In one such investigation[49, 56] involving a study of the progressive histological and biochemical changes occurring prior to and at the time of necrosis, we noted that neutral fat and cholesteryl esters accumulated in the liver, primarily around the central vein areas prior to the development of necrosis. Free cholesterol, phospholipid, and glycogen remained essentially unchanged during this period. Livers showing massive necrosis differed from the non-necrotic livers obtained from animals killed after the same interval on the test diet by a striking increase in free cholesterol and equally striking decreases in phospholipid and glycogen. Subsequently[57] similar analyses were performed on groups receiving the basal necrogenic diet supplemented by one of the following substances: cystine, alpha-tocopherol, and choline chloride. Although the supplement of 0.625% choline chloride kept the level of liver lipids well below that seen in the group on the unsupplemented diet the incidence and time of necrosis were essentially identical. The cystine supplement caused an increase of liver lipid levels but no necrosis occurred and the addition of alpha-tocopherol, also effective in preventing the hepatic lesion, led to an intermediate level of fat. These results indicated that there was no discernible correlation between total liver lipid levels and the development of massive hepatic necrosis. A similar conclusion may be applied to the behaviour of the cholesteryl ester fraction, except that the necrotic livers were always characterized by increased concentrations of this lipid. Free cholesterol levels remained remarkably constant no matter what diet was fed so long as the liver was not the site of acute damage, at which stage a striking increase occurred. Thus although there appeared to be no change in the liver lipid levels that could be used as an indication that necrosis would subsequently supervene, profound changes always occurred in levels of phospholipid, free cholesterol and cholesteryl esters at the time of necrosis.

A finding that may be of great significance was reported by Leaf and Neuberger[65] who noted that on diets low in methionine and cystine the glutathione level in the liver fell markedly but could be restored by supplementation with either of the sulphur-containing amino acids. However, despite the fact that their diets were designed to produce liver necrosis no lesions developed. The writer would like to suggest that the most likely reason for this circumstance was the extremely low level of dietary protein the

effect of which has been previously noted.[7, 42] Dent[66] determined the methionine and cystine levels in the liver protein from rats on diets low in these compounds. He concluded ". . . that the development of acute necrosis is not related to a change in the proportion or amount of these amino acids in liver protein".

We[58] noted that conditions associated with low levels of plasma pseudo-cholinesterase potentiated the development of dietary liver necrosis and conversely those associated with high levels of this enzyme retarded the development of the liver lesion. In the hope that the determination of plasma pseudo-cholinesterase might make possible an earlier assessment of the effects of dietary alterations on the development of hepatic necrosis, the activity of this enzyme was followed in two groups of rats on a basal necrogenic diet, one of the groups being supplemented with alpha-tocopherol. Although in the period prior to the development of acute liver damage there was a significant difference between the two groups, unfortunately, due to the relatively large variation in the plasma levels of the enzyme, the information could not be utilized to predict at an early date in a feeding experiment whether or not an animal is on a diet that would eventually cause acute massive liver necrosis. Greenberg and Hoffbauer[59] followed the excretion of urinary and fæcal coproporphyrin in rats on a basal necrogenic diet but no significant increase such as one would observe in the case of the common hepatoxins was observed.

Perhaps the nearest approach to a test that might enable one to predict the onset of massive liver necrosis is the dialuric acid test described by Rose and Gyorgy.[60] These workers found that within 3 to 7 days of placing rats on a basal necrogenic diet, treatment of the erythrocytes with dialuric acid resulted in hæmolysis. In their experience no rat has ever developed liver necrosis in which this test was not positive. On the other hand, it does not follow that a rat whose red blood cells are hæmolysed by the addition of dialuric acid will inevitably develop acute liver damage. The test is therefore limited in its usefulness.

Olson and Dinning[61, 62] have investigated the behaviour of a number of enzyme systems in the liver of rats receiving a basal necrogenic diet with and without supplements of alpha-tocopherol, methionine, and cystine. They found that whereas succinoxidase and transaminase were essentially normal in all groups, pyruvic acid oxidase was markedly depressed in livers showing necrosis, reduced in livers from animals on the basal diet not showing necrosis and essentially normal in livers from animals receiving supplements of the sulphur-containing amino acids or of alpha-tocopherol. They also found that the content of coenzyme A correlated inversely with the degree of hepatic necrosis. Since coenzyme A contains a thioethanolamine residue they have suggested that this system may be the metabolic link between alpha-tocopherol and the sulphur-containing amino acids. One feature that does not fit adequately into this picture, as has been previously pointed out by the writer, is the relative ineffectiveness of cysteine, presumably an intermediary in the formation of thioethanolamine.

The possibility that alpha-tocopherol might be acting by virtue of its anti-oxidant capacity has interested a number of investigators. In 1950 we tested hydroquinone at a level of 0.1% in a basal necrogenic diet but it had no effect on the development of liver necrosis. On the other hand, Dam and Granados[43] have reported that 0.126% of methylene blue prevented acute hepatic damage in rats fed a basal diet that caused an 80% incidence of acute hepatic damage. They had been led to test the effect of methylene blue because in previous experiments they had found it prevented the appearance of certain other signs of alpha-tocopherol deficiency such as exudation and encephalomalacia in chicks, peroxidation and coloration of depot fat in rats and against hæmolysis of rat erythrocytes with dialuric acid, an effect which could not be reproduced by Heard, Moore and Sharman.[68] So far as the author is aware the prophylactic action of methylene blue has not yet been confirmed. The suggestion made by Dam and Granados[43] that alpha-tocopherol protects against massive hepatic necrosis and lung hæmorrhage, a complication first noted by Hove, Copeland, and Salmon[63] in 1949, by virtue of its anti-oxidant effect is not supported by the fact that alpha-tocopherol quinone was shown by us[15] and by Selzer, Parker, McKenzie and Linder[64] to have some prophylactic action. Furthermore, the latter workers found both the gamma and delta analogues of tocopherol to be ineffective, and this despite the fact that their anti-oxidant activity is greater than that of the alpha form.

In conclusion one can say without equivoca-

tion that although a good deal of information has been uncovered on factors that modify the production of acute massive liver necrosis, the exact sequence of metabolic events leading to the production of this lesion remains to be elucidated.

Although the clinical course and histological picture seen in acute dietary hepatic necrosis is analogous to that seen in acute yellow atrophy in humans, it is impossible to say, at the present time at least, that there is any connection between them. It is quite likely, however, that investigations leading to a better understanding of acute liver injury in experimental animals should at the same time lead to a better understanding of the sequence of events in acute liver injury in humans and point the way to adequate preventive and prophylactic measures.

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RÉSUMÉ

La nécrose aiguë du foie est la plus tragique et la plus rapidement fatale de toutes les nombreuses lésions expérimentalement causées par la mauvaise alimentation. L'importance des expériences de Weichselbaum sur les rats en 1935 réside en ce qu'il produisit des lésions mortelles du foie en soumettant ses animaux à un régime généralement pauvre en protéine et tout particulièrement en méthionine et cystine. Gyorgy et Goldblatt crurent que la carence d'un des facteurs de la vitamine B en était responsable et que la nécrose n'était qu'une phase intermediaire vers l'évolution de la cirrhose. Daft, Sebrell, et Lillie remarquèrent que la choline et la méthionine empêchaient d'infiltration graisseuse et la cirrhose alors que la cystine l'augmentait; que la méthionine ou la cystine prévenaient l'apparition de la nécrose alors que la choline restait sans effet. Hock et Fink rapportèrent que l'intensité de la nécrose produite par un régime à la levure était en rapport direct avec la proportion d'azote et de soufre que contenaient ces levures. Il semble cependant y avoir un principe nécrogène dans la fraction lipidique des levures. La nécrose du foie peut également être causée par l'introduction dans le régime de caséine traitée aux alcalins. Cette lésion répond au traitement à la xanthine et à l'α-tocophérol (Schwarz 1944). L'auteur fait remarquer que de tous les régimes causant la nécrose les plus actifs sont ceux dont les lipides comportent le moins d'α-tocophérol.

Dans les travaux sur les rats, il ressort qu'avec les régimes appropriés, les lésions hépatiques se produisent d'abord chez les petits animaux et seulement que plus tard chez les plus gros, et que les femelles survivent plus longtemps que les mâles. Gyorgy et Schwarz remarquèrent que la Cortisone améliore l'effet nécrogène des diètes de base et que l'extrait thyroïdien accélère considérablement l'apparition des lésions hépatiques aiguës. La cystine ou l'α-tocophérol augmentent la résistance des rats thyrotoxiques. Les régimes deviennent beaucoup plus nécrogènes s'ils contiennent 2% d'huile de foie de

morue. L'addition d'une quantité équivalente de lard contenant des vitamines "A" et "D" sous forme hydrosoluble rend le régime moins nocif. La Vitamine K retarde l'apparition des lésions et B_{12} semble n'avoir aucun effet. Aucune lésion ne résulte d'un régime contenant de 1 à 2%, ou plus de 4% de caséine; toutefois la nécrose hépatique peut être produite avec une teneur de 3 à 4% de caséine dans l'alimentation. Des séparations fractionnées cherchant à isoler le principe nécrogène du lard n'ont abouti qu'à des résultats variables qui cependant ont une certaine ressemblance avec ceux obtenus par l'emploi de l'auréomycine comme agent antinécrogène. Il n'est pas impossible que les résultats dans les deux cas reflètent les variations de la flore intestinale et de ses influences sur l'aborption des aliments dans l'intestin. En dépit du manque de ressemblance de leur structure moléculaire l'α-tocophérol et les amino-acides soufrés ont des effets identiques, qu'ils soient administrés par voie orale ou parentérale. Par contre, la cystéine s'est avérée pratiquement inutile dans la prévention de la nécrose du foie. Les changements cliniques qui accompagnent cette lésion se succèdent avec une rapidité étonnante. Lorsque le stage de la nécrose est atteint, les changements biochimiques les plus marqués se retrouvent au niveau des phospholipides, du cholestérol et de ses esters. Les variations du taux de glutathione du foie, de la pseudocholinesterase du plasma ou de l'excrétion de la coproporphyrine dans les fèces et les urines furent étudiées dans le but d'obtenir des indications pronostiques, mais sans fournir grand résultat. L'épreuve à l'acide dialurique décrite par Rose et Gyorgy n'offre que des possibilités plutôt restreintes. L'immunité que confère l'α-tocophérol ne provient pas de son action antioxidante. M.R.D.

THE ANTEMETIC ACTION OF CHLORPROMAZINE HYDROCHLORIDE*

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THE PURPOSE of this report is to describe the results of an investigation in the Department of Pharmacology at Queen's University of the antemetic action of chlorpromazine hydrochloride. The study of chlorpromazine hydrochloride was part of a program of screening compounds for antemetic activity, which has the objective of finding an antemetic agent that is free of undesired side reactions in a dosage range which effectively prevents or stops vomiting. There is need for such a drug. The general plan of our screening program is to test compounds for their ability to prevent apomorphine-induced vomiting in dogs. The antemetic research program was started in these laboratories at Queen's University in 1950 and is described in another report.[6]

In November of 1951, Professor R. Paul of the Faculté libre des Sciences d'Angers visited these laboratories at Kingston. He described experiments which had been conducted in the department of Direction Scientifique of the Société des Usines Chimiques Rhône-Poulenc at Paris on the ability to potentiate the action of sedatives of a compound which he referred to as 4560-R.P. Professor Paul arranged to have some of this material sent to us. In the *texte manuscrit* of a lecture upon compound 4560-R.P. given by Professor Paul at Montreal and Quebec earlier in November, 1951, this experimental compound was described as related chemically to promethazine hydrochloride B.P. 1953. Amongst his pharmacological studies of 4560-R.P., Professor Paul described its ability to inhibit apomorphine-induced vomiting in dogs. In view of this and of the chemical relationship of chlorpromazine hydrochloride to promethazine hydrochloride, B.P. 1953, or Phenergan hydrochloride, which has been reported previously from this department at Queen's University to have limited antemetic properties,[6] it was decided to run a series of experiments upon chlorpromazine hydrochloride. A preliminary report of our results was presented in abstract to the American Society for Pharmacology and Experimental Therapeutics at the annual spring convention in Chicago, April, 1953.[7]

PHYSICO-CHEMICAL PROPERTIES AND PREPARATIONS

Chlorpromazine hydrochloride is a generic name for the compound described chemically as the hydrochloride of (dimethylamino-l-n-propyl-3)-N-(2-chloro)-phenothiazine. As indicated in the chemical name and in its structural relationships, it is basically a derivative of phenothiazine. It is a light powder, dull white in appearance, practically odourless and having an insipidly bitter taste. It is quite soluble in water forming a solution with a slightly acid reaction. It has a burning and then a prolonged local anæsthetic effect on the tongue. In Canada, the drug has been made available recently by Poulenc Limited of Montreal under the trade name Largactil.* Largactil is dispensed in the form of tablets, each containing 25 mgm. of the drug, and of

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*In the United States of America, it is now available through Smith, Kline and French Laboratories of Philadelphia.

ampoules for intramuscular injection, each containing 10 or 50 mgm. per 2 ml.

ABSORPTION AND FATE

From evidence obtained in these laboratories at Queen's University, as cited below, chlorpromazine may be concluded to be readily absorbed when given by mouth. An oral dose corresponding to 0.5 mgm. per kgm. body weight was metabolized and destroyed in the body of dogs, to the extent that its pharmacologically measured effects had disappeared, by the end of 6 hours. Larger doses were destroyed in the body much more slowly, some effects lasting for several days. When given orally, its pharmacological effects appeared within 1 hour. When given by intramuscular injection, its pharmacological effects became apparent within a minute or two. The intramuscular injection of doses up to 2 mgm. per kgm. body weight produced no local induration nor ulcer formation at the site of injection, such as that reported from injection of similar compounds by Baird and Boyd.[2, 3]

PHARMACOLOGICAL ACTIONS

Apart from its antemetic action, chlorpromazine hydrochloride has other pharmacological effects which may be noted briefly, as seen during the course of our investigations and as noted by others.[13, 14] When used solely as an antemetic agent, these other effects would be considered as untoward or side reactions which would limit the usefulness of the drug. They are as follows:

1. Sedation without hypnosis is a characteristic effect of large doses and appears to a limited degree with doses corresponding to the usual therapeutic dose. A pronounced sedative effect was noted in our dogs, and was confirmed in cats, given oral doses of 10 mgm. per kgm. body weight and over. Less obvious sedation could be determined by measurement, after doses of 1 mgm. per kgm. body weight and over. Obvious sedation appeared as a marked lethargy, no sleeping, and a staggering gait when locomotion was attempted or promoted. Fighting was more common amongst the dogs, suggesting loss of cerebral inhibitions. At the April, 1953, convention of the Canadian Anæsthetists' Society, Ontario Division, at Niagara Falls, and using chlorpromazine hydrochloride as a pre-anæsthetic sedative, Dr. F. Hudon of Quebec described its effect as that of a pharmacological lobotomy.[10] These results suggest that chlorpromazine may have an addiction potential and that excessive use over a long period might lead to serious chronic poisoning of the brain. It has been found to prolong the hypnotic and anæsthetic effects of alcohol, ether and barbiturates, the analgesic effect of narcotics, the spinal cord relaxant effect of mephenesin and to lower body temperature.[13] European clinicians have reported application of these pharmacological effects in the fields of anæsthesiology (*e.g.*[9]), obstetrics (*e.g.*[12]), psychiatry (*e.g.*[1]), and surgery (*e.g.*[11]).

2. Adrenolytic activity has been found to a moderate degree after administration of chlorpromazine hydrochloride.[13] We have not investigated this action, but have noted a bradycardia in dogs following intramuscular injection of 2 mgm. per kgm. body weight. Larger doses of the drug produced respiratory phasic sinus arrhythmia in our animals.

3. Antihistaminic activity has been found to a limited degree after administration of chlorpromazine hydrochloride.[13, 14]

4. Antemetic activity was found to a pronounced degree in dogs following administration of chlorpromazine hydrochloride by Paul[14] and confirmed by Boyd, Cassell and Boyd.[7] Dr. William L. Long[13] has reported to us that investigators in the Smith, Kline and French Laboratories at Philadelphia have also confirmed the antemetic action reported by Boyd, Cassell and Boyd[7] in dogs. Dr. H. L. Borison[4] stated that experiments in his laboratory at the University of Utah in Salt Lake City yielded results similar to those of our preliminary communication.[7] European investigators have reported a pronounced antemetic action in patients with severe vomiting associated, for example, with pregnancy (*e.g.*[15]).

Toxicology. — We did not systematically investigate the acutely lethal dose of chlorpromazine hydrochloride but found that oral administration of 60 mgm. per kgm. to dogs did not kill any of our animals. This dose left the dogs weak, listless and inco-ordinate in locomotion for 2 to 3 days. The possibility of chronic cerebral poisoning and addiction has been noted above.

Antemetic screening. — Chlorpromazine hydrochloride was screened for antemetic activity after the method of Boyd and Boyd.[8] The dogs used have been trained for antemetic studies and have developed no chronic tolerance toward apomorphine hydrochloride during the past three to

four years. They were housed in the animal quarters of the Department of Pharmacology at Queen's University and fed bread, milk, meat, Purina Fox Chow Checkers, vitamin supplements and water *ad libitum*. They have remained in good health and have maintained body weight throughout these experiments. They were given no food for 16 hours previous to the antemetic tests which were conducted at 3 to 7 day intervals. The chlorpromazine hydrochloride was administered by mouth in capsule form, each dose to 8 dogs which were used in cross-over as controls. One hour later, apomorphine hydrochloride was injected and the effects of chlorpromazine hydrochloride upon the vomiting syndrome are summarized in Table I.

TABLE I.

THE EFFECT OF ORAL ADMINISTRATION OF CHLORPROMAZINE HYDROCHLORIDE, ONE HOUR BEFORE INTRAMUSCULAR INJECTION OF 0.05 MGM. PER KGM. BODY WEIGHT OF APOMORPHINE HYDROCHLORIDE, UPON THE SUBSEQUENT VOMITING SYNDROME IN DOGS (8 ANIMALS PER DOSE OF CHLORPROMAZINE HYDROCHLORIDE).

Dose of chlorpromazine hydrochloride (mgm. per kgm. body weight)	*Incidence of vomiting*	*Mean frequency of vomiting*	*Minutes (mean) before onset of bomiting*	*Mean freqency of retching*	*Mean index of physical activity*
0.0	100%	4.8	6	45	2.4
0.0	100%	4.4	6	39	2.3
0.0	100%	5.7	6	30	2.2
0.5	100%	3.8	6	34	2.1
1.0	100%	2.9	7	27	2.2
2.0	63%	2.4	8	14	1.8
5.0	13%	0.5	6	4	1.6
10.0	13%	0.2	6	1	2.1
20.0	0%	0.0	–	0	2.2
40.0	0%	0.0	–	0	1.2
60.0	0%	0.0	–	0	1.8

In this series of experiments, the frequency of vomiting and retching was lessened by an oral dose of 1 mgm. per kgm. body weight of chlorpromazine hydrochloride. Complete suppression of vomiting and retching occurred following administration of a dose of 20 mgm. per kgm. body weight. The interval between the time of injection of apomorphine hydrochloride and the time of onset of vomiting was not affected by administration of chlorpromazine hydrochloride. The larger doses of chlorpromazine hydrochloride caused a decline in the mean index of physical activity. The degree of antemetic action seen in this series of experiments was more pronounced than that produced by hyoscine hydrobromide[8] or by antihistaminic agents so far screened by this technique.[6] It corresponded to that which we have been able to produce otherwise only by complete anæsthesia of the emetic reflex.

Limited emetic challenge. – The degree of emetic challenge presented to the animals in the series of experiments described above was a considerable one, in that all of the untreated control dogs vomited. In an epidemic of say motion sickness, vomiting amongst all exposed persons is of rare occurrence. Therefore the effect of chlorpromazine hydrochloride upon the lesser emetic challenge of injection of 0.025 and 0.0125 mgm. per kgm. body weight of apomorphine hydrochloride was tried. The results have been summarized in Table II.

In this series of experiments, chlorpromazine hydrochloride completely suppressed vomiting from injection of 0.025 mgm. per kgm. body weight of apomorphine hydrochloride in a dose of 2 mgm. per kgm. body weight. Vomiting occurred in 50% of dogs following injection of 0.0125 mgm. per kgm. body weight of apomorphine hydrochloride and was completely suppressed by previous oral administration of 1 mgm. per kgm. body weight of chlorpromazine hydrochloride. The index of physical activity declined after administration of all doses of chlorpromazine hydrochloride which, it should be noted,

TABLE II.

THE EFFECT OF ORAL ADMINISTRATION OF CHLORPROMAZINE HYDROCHLORIDE TWO HOURS BEFORE INTRAMUSCULAR INJECTION OF 0.025 AND 0.0125 MGM. PER KGM. BODY WEIGHT OF APOMORPHINE HYDROCHLORIDE UPON THE SUBSEQUENT VOMITING SYNDROME IN DOGS (6 ANIMALS PER DOSE OF CHLORPROMAZINE HYDROCHLORIDE).

Dose of chlorpromazine hydrochloride (mgm. per kgm. body weight)	*Incidence of vomiting*	*Mean frequency of vomiting*	*Mean frequency of retching*	*Mean index of physical activity*
After 0.025 mgm. per kgm. of apomorphine hydrochloride				
0.0	83%	3.8	32	1.7
0.5	83%	2.9	21	1.6
1.0	50%	1.0	11	1.5
2.0	0%	0.0	0	1.1
5.0	17%	0.2	1	1.3
10.0	0%	0.0	0	0.9
After 0.0125 mgm. per kgm. of apomorphine hydrochloride				
0.0	50%	0.8	9	2.0
0.5	33%	0.5	4	1.8
1.0	0%	0.0	0	1.9
2.0	0%	0.0	0	1.6
5.0	0%	0.0	0	1.7
10.0	0%	0.0	0	1.7

TABLE III.

THE EFFECT OF INTRAMUSCULAR INJECTION OF CHLORPROMAZINE HYDROCHLORIDE, 15 SECONDS AFTER INTRAMUSCULAR INJECTIONS OF 0.05 MGM. PER KGM. BODY WEIGHT OF APOMORPHINE HYDROCHLORIDE, UPON THE SUBSEQUENT VOMITING SYNDROME IN DOGS (6 ANIMALS PER DOSE OF CHLORPROMAZINE HYDROCHLORIDE).

Dose of chlorpromazine hydrochloride—mgm. per kgm. body weight	0.0	0.5	1.0
Incidence of vomiting	100%	50%	17%
Mean frequency of vomiting	4.5	1.0	0.2
Mean frequency of retching	38	11	4
Mean index of physical activity	2.0	1.7	1.7

were given 2 hours before injection of apomorphine hydrochloride in this series of experiments.

Onset of antemetic action.—The next point investigated was how rapidly could an intramuscular injection of chlorpromazine hydrochloride produce an antemetic action toward apomorphine hydrochloride. To investigate this, chlorpromazine hydrochloride was injected intramuscularly immediately (within 15 seconds) after intramuscular injection of apomorphine hydrochloride. The results obtained are summarized in Table III.

Within 5 to 8 minutes after injection of 0.05 mgm. per kgm. body weight of apomorphine hydrochloride, vomiting ordinarily begins. The injection of 0.5 mgm. per kgm. body weight of chlorpromazine hydrochloride reduced the incidence and frequency of vomiting and retching. The injection of 1 mgm. per kgm. body weight of chlorpromazine hydrochloride almost completely suppressed the vomiting syndrome. Intramuscular injection of chlorpromazine hydrochloride was quickly followed by its antemetic action, in the matter of a few minutes.

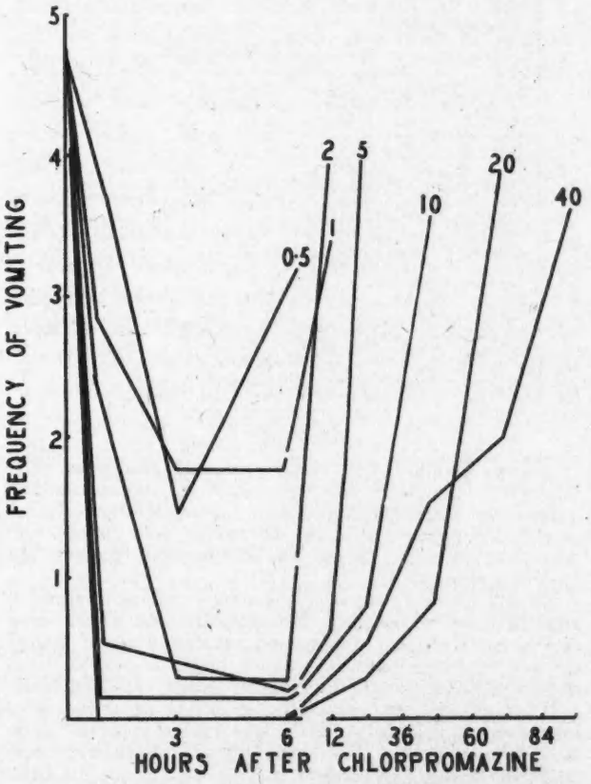

Fig. 1.—Duration of the ability of chlorpromazine hydrochloride to inhibit apomorphine-induced vomiting spells in dogs. The dose of chlorpromazine hydrochloride in mgm. per kgm. body weight by mouth, is indicated at the right end of each graph line.

Duration of antemetic action.—To find the duration of antemetic action, groups of 6 dogs each were given by mouth from 0.5 to 40 mgm. per kgm. body weight of chlorpromazine hydrochloride, and challenged with intramuscular injections of 0.05 mgm. per kgm. body weight of apomorphine hydrochloride, at intervals of 1, 3, 6, 12, 24, 48, 72, 96, etc., hours afterward, until the vomiting syndrome had been restored. The results have been charted in Fig. 1, by illustrating changes at these intervals in the frequency of post-apomorphine emetic spells.

The durations of antemetic action of oral chlorpromazine hydrochloride were found to be as follows:

From 0.5 mgm. per kgm. body weight...	3 to 6 hours
From 1.0 mgm. per kgm. body weight...	6 to 12 hours
From 2.0 mgm. per kgm. body weight...	9 to 12 hours
From 5.0 mgm. per kgm. body weight...	18 to 24 hours
From 10.0 mgm. per kgm. body weight...	24 to 36 hours
From 20.0 mgm. per kgm. body weight...	60 to 72 hours
From 40.0 mgm. per kgm. body weight...	72 to 96 hours

Curative antemetic action.—The previous series of experiments have demonstrated that chlorpromazine hydrochloride is capable of preventing apomorphine-induced vomiting. The next experiments were designed to investigate the ability of chlorpromazine hydrochloride to stop apomorphine-induced vomiting after it had begun. The beautiful experiments of Professor H. L. Borison of Salt Lake City, Utah, demonstrating a chemoreceptor trigger zone in the medulla oblongata as the site of emetic action of apomorphine hydrochloride, have already been reviewed by one of us.[5] Immediately after the occurrence of the first apomorphine-induced vomiting spell, dogs were injected intramuscularly with chlorpromazine hydrochloride.

TABLE IV.

THE EFFECT OF INTRAMUSCULAR INJECTION OF CHLORPROMAZINE HYDROCHLORIDE IMMEDIATELY AFTER OCCURRENCE OF THE FIRST VOMITING SPELL, INDUCED BY INTRAMUSCULAR INJECTION OF 0.05 MGM. PER KGM. BODY WEIGHT OF APOMORPHINE HYDROCHLORIDE, UPON THE SUBSEQUENT VOMITING SYNDROME OF DOGS (6 ANIMALS PER DOSE OF CHLORPROMAZINE HYDROCHLORIDE).

Dose of chlorpromazine hydrochloride—mgm. per kgm. body weight	0.0	1.0	2.0
Incidence of vomiting	100%	67%	50%
Mean frequency of vomiting	2.9	1.0	0.5
Mean frequency of retching	27	7	5
Mean index of physical activity	1.8	2.2	1.0

The results obtained are summarized in Table IV. It may be seen that administration of chlorpromazine hydrochloride in doses of 1 and 2 mgm. per kgm. body weight intramuscularly

decreased the incidence and frequency of subsequent vomiting and retching. The larger dose of chlorpromazine hydrochloride again decreased physical activity.

Species variation.—The experiments described above have demonstrated that chlorpromazine hydrochloride quickly and for a fairly prolonged period prevents and suppresses apomorphine-induced vomiting in dogs. The dog is the only readily available laboratory animal which vomits with the same ease as does man. The cat can be made to vomit after injection of apomorphine hydrochloride but is resistant to its emetic action. Doses of apomorphine hydrochloride of the order of 20 mgm. per kgm. body weight are required to produce vomiting in cats and these doses also produce maniacal excitement. It was decided to try the antemetic action of chlorpromazine hydrochloride upon cats and 10 animals were given 20 mgm. per kgm. body weight by mouth 1 hour before intramuscular injection of 20 mgm. per kgm. body weight of apomorphine hydrochloride. There followed a slight decrease in the incidence and mean frequency of vomiting and retching with reduction by some 50% of the mean index of physical activity. The antemetic action was not nearly as pronounced in cats as it was in dogs.

SUMMARY

The oral or intramuscular injection of chlorpromazine hydrochloride (4560-R.P., Largactil), in doses approximating the usual human therapeutic dose, effectively inhibited and in many instances completely suppressed, apomorphine-induced vomiting in dogs.

Chlorpromazine hydrochloride was readily absorbed from the gastrointestinal tract and exerted its antemetic effect quickly, particularly after intramuscular injection. The antemetic action of lower doses lasted several hours and of higher doses several days.

A sedative side reaction appeared to a pronounced degree following administration of large doses of chlorpromazine hydrochloride and to a lesser degree following lower doses of the drug. Large doses produced a respiratory phasic sinus arrhythmia.

The authors wish to acknowledge the receipt of supplies of chlorpromazine hydrochloride from Poulenc Limited of Montreal through the courtesy of Mr. A. Manchec.

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RÉSUMÉ

L'administration de chlorhydrate de chlorpromazine (4560-R.P. Largactil) par voie orale ou intramusculaire à dose se rapprochant de la dose employée en thérapeutique humaine, diminue, et même dans certains cas supprime chez les chiens les vomissements causés par l'apomorphine.

Le chlorhydrate de chlorpromazine est promptement absorbé dans le système gastro-intestinal et déploie son action antémétique rapidement, surtout lorsque donné en injection intramusculaire. Ses effets à petites doses durent plusieurs heures, et à hautes doses, plusieurs jours.

Il manifeste des propriétés sédatives très marquées lorsqu'employé à hautes doses, et moins marquées losque la dose est réduite. De fortes doses provoquent une arythmie sinusale respiratoire. M.R.D.

THE CARDIOVASCULAR ADJUSTMENTS IN ANÆMIA: A REVIEW*

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THE CARDIOVASCULAR adjustments which occur in anæmia are of interest to those working in the field of the physiology of anoxia and to those who encounter this condition in medical practice. Although these adjustments have been well described, less is known about the time relationships of the changes as the disease progresses in severity and as a cure is effected by various means. Further, the mechanism responsible for the compensatory elevation in cardiac output and other vascular changes regularly observed in anæmia have not been clarified.

The purpose of the present paper is to discuss the cardiovascular adjustments found in anæmia in the light of recent investigation directed at determining the sequential relationship of the

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various changes and the mechanisms responsible for them.

General considerations. – Normally arterial blood contains, in combination with hæmoglobin, approximately 20 c.c. oxygen/100 c.c. blood and the arterial hæmoglobin is 95 to 97% saturated with oxygen. The blood as it flows through the capillaries gives up an average of 5 c.c. of oxygen/100 c.c. blood (arteriovenous oxygen difference) so that the mixed venous blood in the right heart contains 15 c.c. of oxygen/100 c.c. blood. Thus the hæmoglobin of mixed venous blood is about 70% saturated and the percentage utilization of arterial oxygen (the ratio, expressed in per cent, of the amount abstracted by the tissues to the amount present in arterial blood) is approximately 25%.

The essential characteristics of anæmic anoxia,[1] uncomplicated by other factors, which differentiate it from other forms of anoxia are a decreased oxygen content of arterial blood paralleling the reduced hæmoglobin level, whereas the hæmoglobin present in arterial blood has a normal oxygen saturation.[2 to 5]

Under the conditions imposed by the anæmic state the tissue oxygen supply is maintained by two mechanisms which may act singly or together. These mechanisms are (1) an increased cardiac output[2, 4, 7 to 14] which effects an increased delivery of oxygen to the tissues, and (2) a greater percentage utilization of the oxygen available to the tissues.[2, 4, 6, 10 to 14]

Cardiac output and oxygen utilization.—Some controversy exists in respect to the degree of anæmia necessary before an elevation in cardiac output occurs. Some investigators find no increase in cardiac output until the hæmoglobin level is reduced to approximately 7 gm./100 c.c. blood. With lower hæmoglobin values marked elevations in cardiac output regularly occur.[9, 11 to 16]

An increase in cardiac output may be noted at levels of hæmoglobin above 7 gm./100 c.c blood especially when the anæmia occurs rapidly as is the case following a severe hæmorrhage.[11, 13, 17] In these circumstances the elevation in cardiac output may appear as soon as 4 to 6 hours after hæmorrhage.[17]

At reduced hæmoglobin levels and prior to an elevation in cardiac output, the usual abstraction of 5 c.c. of oxygen/100 c.c. blood by the tissues means that the percentage utilization of arterial oxygen is increased. Under these conditions the venous blood is less saturated with oxygen and tissue oxygen tension falls. This method of tissue oxygen supply apparently may continue singly until, as previously indicated, a critical although variable hæmoglobin level is reached at which time the cardiac output increases.

The elevation in cardiac output which is characteristic of severe anæmia is associated with a decreased arteriovenous oxygen difference.[2, 4, 7, 10, 12 to 15] Little, if any, of the increase in cardiac output in uncomplicated anæmia can be accounted for on the basis of increased total body oxygen consumption.[4, 6, 7, 10, 11, 12, 14, 18] The result is that the oxygen saturation of venous blood and thus tissue-oxygen tension, although still below normal, are increased over that which would obtain if an increase in cardiac output did not occur. Similarly, the increase in percentage utilization of arterial oxygen is less marked than would be observed in the absence of a rise in cardiac output.

Thus it may be seen how the burden of oxygen supply to the tissues is divided, a part taken by an increased cardiac output and part by an increased percentage utilization of available oxygen. Indeed, if the percentage utilization of arterial oxygen is not increased, much higher levels of cardiac output are required to supply the tissues with oxygen.[13, 15]

It has previously been pointed out that the rise in cardiac output cannot be ascribed to an increased metabolic rate, for only slight if any increase in oxygen consumption is observed. However, the metabolic rate can profoundly effect the level of cardiac output. Thus an infection in the presence of severe anæmia may, because of the increased metabolic rate associated with the infection, partially account for, or add to, the rise in cardiac output.[19] Conversely, severe malnutrition may be associated with a sufficient decrease in metabolic activity that a normal cardiac output is adequate to supply tissue oxygen demands.[20, 21]

The fact that tissue oxygen tension, as reflected by the oxygen saturation of venous blood, is low when the cardiac output is elevated has prompted several workers to suggest a balanced relationship between tissue oxygen tension and cardiac output.[11, 12, 14] According to this concept the increased cardiac output is the result of a decrease in total systemic resistance[12 to 16] which in turn results from the direct effects of anoxia on the peripheral blood vessels.[22]

More recent studies on dogs made anæmic by daily venesections have caused some reason to doubt this theory. In the experiments of Hatcher *et al.*[15, 16] dogs were bled of the order of 100 to 200 c.c. per day for 6 to 10 days, and the cardiovascular system was studied during and after the production of a severe anæmia. Maximum anoxic stress was found at the end of the venesection phase prior to any increase in cardiac output at which time the total systemic resistance was at a maximum. After the final venesection the cardiac output rose gradually over a period of 1 to 7 days during which time the hæmoglobin and blood volume remained unchanged from values noted at the end of the venesection period. The total systemic resistance decreased only as the cardiac output rose, reaching minimum values at the time of the highest cardiac output, when the anoxic stress was at least less marked than was the case at the end of the venesection period. Furthermore, in recent studies by Sereny *et al.*[23] the cardiac output did not return to control levels for several hours following rapid correction of the anæmia by an infusion of concentrated whole blood. In these studies, despite relief of the anoxic stress, the total systemic resistance remained low and increased only as the cardiac output decreased toward control levels.

Thus it would appear that although tissue oxygen tension may well be an initiating stimulus for the rise in cardiac output it would not seem to be a sustaining factor. These findings further suggest that the low total peripheral resistance observed in association with the high cardiac output in anæmia is not the result of anoxia acting on peripheral vessels but rather represents a homeostatic mechanism which maintains a near normal (or slightly decreased) blood pressure[7, 10, 13 to 17] during the period of elevation in cardiac output.

Peripheral circulation.—The decrease in total systemic resistance observed during the high cardiac output phase implies a peripheral vasodilatation but the evidence indicates that this is not a generalized response.

The rate of blood flow in the forearm (muscle blood flow) is regularly increased in anæmia[24] but the hand (skin) blood flow is reduced.[24, 25, 26] Plethysmographic studies of the peripheral circulation in dogs also indicate an increased muscle blood flow[15] but the paw (skin) blood flow is variable.[15] A decreased rate of blood flow to the kidneys is described which is largely the result of an afferent arteriolar vasoconstriction.[27] The cerebral blood flow as studied by the nitrous oxide technique is increased in anæmia.[28]

Blood volume and venous filling pressure.—The total blood volume is decreased in anæmia[13, 15, 21, 29, 30] which may be due to a decrease in red blood cell volume only.[29]

Sharpey-Schafer[13] described a reduced total blood volume, high cardiac output, and elevated right atrial pressure in anæmic patients. On this evidence he postulates the existence of a venomotor mechanism which decreases the capacity of the vascular bed and increases the venous filling pressure. Sharpey-Schafer considers that the elevated filling pressure under these circumstances, acts in accord with Wigger's modification of Starling's law to determine the level of cardiac output. Brannon *et al.*[14] have been unable to corroborate the finding of a raised right atrial pressure except in the presence of congestive failure.

However, Hatcher *et al.*[15, 16] working with dogs, demonstrated a normal or elevated filling pressure during the high cardiac output period. In this investigation it was possible to study the progressive development of these changes. In most of the animals, elevations in right atrial pressure were observed as much as three days before the rise in cardiac output was initiated. If the elevation in right atrial pressure were the cause of the elevation in cardiac output as suggested by Sharpey-Schafer[13] then one might expect the rise in output coincident with the initial rise in right atrial pressure which, in fact, was not observed for several days.

It is possible that the maintenance of a normal or elevated right atrial pressure in anæmia represents, as Sharpey-Schafer pointed out, an increase in venomotor tone with a shift of blood to the central veins and heart. But the evidence obtained from anæmic dogs as well as collateral evidence from infusion studies[13] suggests that the elevated right atrial pressure is not the cause of the elevated cardiac output but rather may represent an adaptation for the decrease in blood volume. Such an adjustment would serve to maintain cardiac filling at the best possible level.

Mechanisms.—Certain possible mechanisms to account for the rise in cardiac output have already been outlined. The first is the concept of a balanced relationship between tissue oxygen

tension and cardiac output. Some of the main criticisms of this theory have been presented. The second concept concerns the rôle of the increased filling pressure. This adjustment although important cannot be considered a cause for the increased cardiac output on the basis of the evidence given.

A third possibility remains. Sharpey-Schafer has commented on the slow rise in cardiac output to maximum values following hæmorrhage and suggests that a slow-acting or gradually elaborated humoral mechanism may be involved in this change.[17]

In the posthæmorrhagic anæmia studies of Hatcher *et al.*[15, 16] the rise in cardiac output, once initiated, continued to peak values over a period of 1 to 7 days independent of further change in hæmoglobin and blood volume. It has further been shown that the cardiac output does not return to normal for several hours following rapid correction of the anæmia by a concentrated whole blood infusion. The slow rise in cardiac output in response to anæmia, as well as its slow disappearance even on rapid correction of the anæmia, is in agreement with the thesis that there is a humoral mechanism involved.

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A CENTURY OF PSYCHIATRIC TEACHING AT ROCKWOOD HOSPITAL, KINGSTON*

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WITH THE ESTABLISHMENT of a school of medicine at Queen's University in 1854, it is of interest to note that even at that early date, some facilities for the teaching of psychiatry were not only available but were being utilized. Certainly, however, they were limited to a small number of criminally insane patients who were being cared for in the basement of the Kingston Penitentiary. A year later, no doubt as a result of pressure on the part of the medical profession and the public generally, the Dominion Government purchased the Cartwright estate, just west of Kingston, and on this site erected Rockwood Hospital.[1] Throughout the years, this institution has had a very close association with Queen's Medical School, and as well as being utilized for the teaching of mental disorders has also provided much clinical material in the fields of medicine, gynæcology, surgery and pathology.

The first medical superintendent of Rockwood Hospital was Dr. John P. Litchfield who served from 1855 until his death in 1868. He also had the distinction of being one of the original members of Queen's Medical Faculty. The College Calendar[2] of 1854-55 refers to him as follows:

"Lectures in Forensic and State Medicine, Prof. J. P. Litchfield, M.D. The Professor of this branch being Medical Superintendent of the Rockwood Lunatic Asylum, will have ample opportunities of instructing his classes in the important subject of Psychological Medicine. Fee for course—$6.00."

Unfortunately, we have no record of the extent and nature of these lectures.

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Dr. Litchfield's earlier history is somewhat shadowy and according to Gibson[3] there were some doubts among his contemporaries that he had even taken a medical degree. Be that as it may, his ability must have been sufficient to impress the college authorities. As the first teacher of mental disorders, at Queen's his background of experience made him a logical choice. Formerly inspector of hospitals in South Australia, before coming to Canada, he had also served several years as superintendent of the Walker Asylum in England.

Rockwood Hospital was the second mental institution in the province, the first having been built at Toronto a few years earlier. Originally and for thirteen years, the stone stables* on the hospital property used to house twenty-four female patients and it was not until 1868, when the west wing of the main building was completed for women, that the stable asylum was vacated.

Psychiatric teaching in those early days advocated "no restraint", a policy which unfortunately was not generally accepted. All mental disorders were thought to have an organic basis, and indeed, the *American Journal of Insanity* championed this cause. The physical etiology of mental illness became the centre of a controversy which existed among several schools of thought at that time.

Therapeutic procedures around 1850 still recommended blood lettings and the application of leeches in acute mania, with a caution to guard against exhaustion or collapse. Emetics were considered useful in torpid states, as in melancholia with dyspeptic disorder. Sedatives in common use were opium, chloral hydrate and potassium bromide combined with cannabis indicæ. Another sedative of special value adopted a little later and according to Lett[4] "pleasant to take, advantageous in its tonic effects and conducive to sound sleep in violent mania (was) a bottle of the best Scotch Ale or Dublin Stout; a medicine that will bear repetition with the best results and no straight jacket in the world will contribute better to quietness and repose."

Medical journals and newspaper periodicals had been advocating humane and rational treatment for the insane and deplored the use of bodily restraint, coercion and sometimes flagellation. At Rockwood, it was not until 1878 that restraint was gradually abolished. That rest and a quiet hospital atmosphere were important in the treatment of the mentally ill, Dr. John R. Dickson, who succeeded Dr. Litchfield in 1868, was fully aware. During Dr. Dickson's tenure of office, he introduced occupational therapy as well as other valuable reforms, which no doubt, he stressed to his student classes. Some nine years after assuming superintendency, the hospital passed from Dominion to Provincial authority (1877) and at Dr. Dickson's insistence, insane criminals whose sentences had not expired, were transferred back to the penitentiary. Queen's Calendar[5] (1875-76), mentioning the clinical facilities in the two general hospitals of Kingston states, "students can, if they desire, visit the insane asylum with Dr. Dickson". Apparently, psychiatry was not then a compulsory course and no examination was given in the subject.

In 1878 Queen's had her third Professor of Mental Diseases in the person of Dr. W. A. Metcalfe who was appointed superintendent of the Kingston Asylum, following Dr. Dickson's illness and subsequent retirement. Dr. Metcalfe was a man of great promise and had been trained by Dr. Joseph Workmen who at that time was probably Canada's most outstanding alienist. Much that is best in the early system of caring for the insane in Canada may be traced to his influence. Dr. Metcalfe, against strenuous opposition of nursing and attendant staff, abolished restraint which until then was employed at Rockwood. An earnest and capable administrator and an enthusiastic teacher, he laboured for his patients until August 13, 1885, when, while making his usual ward round in company with his assistant, he was fatally stabbed by a criminal lunatic. Dr. C. K. Clarke, who had been assistant, was immediately appointed superintendent and Professor of Mental Disorders, remaining at Rockwood until October, 1905.

A review of Queen's medical examination papers from 1880-1890 does not reveal any set paper on mental disorders, but the jurisprudence examination usually had one or two psychiatric questions. These are of interest in the light of present day knowledge. For example, in 1881 students were asked "What are the legal and medical tests for insanity?" "Diagnose acute mania and explain the medicolegal relations of

*These stables built on the Cartwright Estate and subsequently purchased by the Dominion Government and used as an asylum for women, were responsible for a bit of doggerel at the time of their erection. "Oh would to God that I were able, to build a house like Cartwright's stable. It fills my heart with great remorse, to be worse housed than Cartwright's horse."

delirium tremens and drunkenness"; and in 1886 "Explain as far as you are able what constitutes insanity." "Describe general paralysis of the insane", and in 1888-89 "Distinguish between idiocy and mania". "Describe melancholia." "What are the characteristic symptoms of mania, monomania and melancholia?"; essentially descriptive psychiatry with little or no emphasis on psychodynamics or therapy.

Dr. Clarke, during his superintendency, recognized that non-restraint could only be accomplished along with suitable occupation, instituted hospital industries—mat, brush and broom making. As well as introducing reforms in the hospital, he started the Rockwood Training School for Nurses, one of the first of seven established in America. The training school is still functioning, the 62nd class of graduates completing their course of studies this year. Dr. Clarke, an able teacher, inculcated the spirit of scientific investigation into his staff and Rockwood became a centre of inspiration for the advancement of psychiatry and easily ranked as the most progressive and successful of all the Ontario Institutions.

Dr. John Webster, who was on the Rockwood staff in 1890, makes reference[6] to Dr. Clarke's lectures and clinics which were given during the summer session. "The students attended or not as they felt inclined, the lectures were non-curricular and no examination was held." Dr. Webster recalls surgical and pathological clinics which he himself held and which students were invited to attend. Credit for performing the first lumbar puncture in a case of general paralysis is given to Dr. Webster. On Dr. W. T. Connell's return from England and assuming the professorship of Pathology and Bacteriology at Queen's (1895), he took over the pathological and autopsy service at Rockwood. Clouston's text in mental diseases was generally in use at that time. In the fall of that same year, Queen's Calendar makes reference to Dr. C. K. Clarke giving a course of ten lectures on mental diseases, and a compulsory intermediate examination in medical jurisprudence included toxicology and mental disorders.

When Dr. Clarke left Rockwood in 1905, Dr. Edward Ryan took over the hospital and became Queen's fifth Professor of Mental Diseases. Although without experience in psychiatry, he recognized the Rockwood spirit and wisely determined to keep the scientific side in the foreground, just as his predecessors had done. An added stimulus to psychiatry was given in 1907 when Doctors Ryan, Clarke and the Honourable Dr. Willoughby were appointed a committee by the Ontario Government to go abroad and study the psychiatric hospitals of the Old World. They were particularly impressed with the work of Kraepelin and Alzheimer of the Munich Clinic in Germany. The findings of this committee[7] were published in 1908 in which psychiatric clinics in university centres were strongly recommended. It was not until 1926 that the first such hospital was opened in Toronto.

Under Dr. Ryan's direction Rockwood was continually in the foreground of psychiatric advances and, one can honestly say, he was the inspiration to many a medical student fortunate enough to live under his influence and instruction. He encouraged senior undergraduates to intern at Rockwood, which practice is still continued to this day. As a result, a large number of Queen's graduates chose psychiatry as their life's work and many of them subsequently became superintendents of provincial and state institutions. Dr. Ryan was constantly stressing the great opportunities in the field of mental medicine, and he undoubtedly foresaw the outstanding psychiatric developments of the past quarter century. Unfortunately, from 1900 to 1920, in spite of advances in the clinical fields, psychiatry in medical education moved slowly. There was little place for this science in the old curriculum. The rapid growth of the other biologic and seemingly more strictly medical sciences had been crowding and stretching the curriculum. Hence, though the importance of psychic factors in diseases had been increasingly recognized and the need for psychological investigation had been felt, it was difficult to make a place for psychiatry in the medical course. In 1909 a model medical curriculum[8] formulated by a large group of educators and sponsored by the American Medical Association scarcely mentioned psychiatry in an extensive discussion of the content of the curriculum, and only twelve hours of didactic instruction in the junior year was allotted to psychiatry and neurology together.

In 1922 the course in mental diseases at Queen's consisted of approximately twenty lecture periods lasting one hour followed by a clinical presentation of similar length. A written examination and the completion of one clinical

assignment were requisite for graduation. In recalling psychiatric instruction given in the early twenties, the study of mental disorders was limited entirely to the institution and mental disease was isolated from general medicine. Teaching was largely descriptive, the organic point of view still dominating, and the instruction was given in the final clinical year only. Pessimism as to the outcome of most mental disorders was prevalent which no doubt contributed to what has often been referred to as therapeutic nihilism.

Dr. Ryan remained at Rockwood until his retirement in 1930 to be followed by Dr. T. D. Cumberland, Dr. E. A. Clarke, Dr. C. M. Crawford, Dr. J. S. Stewart and the author. Since 1940 the Department of Psychiatry at Queen's has been under the professorship of Dr. C. H. McCuaig, assisted by the Rockwood medical staff and private practising psychiatrists.

In the early 'thirties, through the impetus of the National Committee for Mental Hygiene and the establishing of the American Board of Psychiatry and Neurology (1934-35), psychiatric education was stepped up. Attitudes of medical schools, and the profession generally, changed. Psychiatry was no longer isolated from other clinical disciplines, there was an acceptance of dynamic principles, teaching became interpretive, relating to the patient in terms of deviations from the normal, psychotherapeutic teachings were developed, therapeutic nihilism was broken down, psychosomatic concepts were accepted and psychiatry was introduced into the preclinical curriculum. At Queen's, from a twenty-hour course of lectures and clinics in the final year, instruction is now given in the last four years and, in all, some 150 hours are devoted to psychiatric teaching. This compares favourably with the United States where the average teaching time in medical schools is approximately 152 hours. As a preparation to a course in psychopathology, which is given during the third year, medical students receive a course in psychology during their second year. In the fourth year lectures and clinics covering the various forms of mental disorders, with special emphasis on psychoneurotic and psychosomatic disorders, are all given at the General Hospital. The fifth year includes lectures, clinics and demonstration of major psychoses and psychoneuroses at both Rockwood and the General Hospital, and at the Sunnyside children's shelter excellent opportunities are afforded for the study of emotionally disturbed children. In the final year, clinical presentations and discussions of community psychiatric problems round out the course.

In addition to developing the instructional program, one of the principal aims of the Department of Psychiatry is to help students mature emotionally during their years in medical school. This aspect of training is not limited to the Psychiatry Department, since it is the function of the whole faculty. When mental health problems do occur in individual students, these are invariably referred to the psychiatrist for therapy.

Thus it will be seen that psychiatry is gradually assuming its rightful place in the medical curriculum. MacCalman[9] says "Medical education despite its increasing materialism has remained a preparation for a life dedicated to social service. Infiltrating psychiatric knowledge into other branches of medicine has progressed to a point that students recognize and accept the part which psychological medicine has to play in the prevention and treatment of ill health." Whereas, but a few years ago, psychiatric concepts and, indeed, psychiatrists were not too well received, there have since been great changes in the attitudes of the general public and even of our own profession. Medical students are now made aware of emotional experiences in health and disease, emotional factors in the relationship between patient and physician and the possibilities of comprehensive medical care. We must expect more and more that the various problems of adjustment will be handled by the internist and the general practitioner rather than by the psychiatrist exclusively. The growing integration of psychiatry with the rest of the curriculum indicates clearly that the general practitioners of the future will be much better fitted to deal with the patient as a whole. At present, emphasis is on the development of psychiatric divisions in general hospitals. (Such a unit of forty beds will soon be added to the Kingston General Hospital and Rockwood is to have a new 500-bed addition which will greatly improve teaching facilities, especially in the hospital residency program leading to specialist certification by the Royal College of Physicians and Surgeons of Canada).

In a hundred years of operation, Queen's has seen many changes. Medical education has de-

veloped vigorously and has kept pace with and assimilated the contributions of science to medicine. Psychiatry has now achieved an important place in the college curriculum. Rockwood Hospital has played a valued rôle in this century of medical progress and, if psychiatric advances of the past twenty-five years are any indication of the future, one can look forward with confidence to even greater achievements.

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A FAMILY OUTBREAK OF LEAD POISONING FROM THE BURNING OF STORAGE BATTERY CASINGS*

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IT IS WELL RECOGNIZED that young children with depraved appetite or pica may chew lead-containing paint from cots, cribs, play-pens, dolls and toys, from furniture, window-sills and verandah railings and so develop symptoms of lead-poisoning over a period of weeks and even months. The poisoning occurs in the large majority of cases between one and three years of age or during the "teething period". According to Huntington Williams,[1] 347 cases of lead poisoning have been diagnosed in Baltimore children between 1931 and 1951 and 60% of these occurred in children in their second year of life, when there is a strong tendency for children to put objects into the mouth. The children lived in old rented houses where lead paint had been used for many years on the frames and sills of windows and where flaking and scaling of the paint had occurred. Investigation revealed in general that the children had chewed the window sills or ingested flakes of paint.

Lead poisoning in older children is more frequently due to other modes of lead absorption, such as accidental ingestion of lead paint in the families of house painters or inhalation of lead fumes from the burning of storage battery boxes. A series of 40 cases of lead poisoning from the burning of discarded battery casings as a source of fuel in squalid, poverty-stricken negro homes in Baltimore was reported by Huntington Williams *et al.*[2] in 1933. Exposure had been intermittent for 6 to 12 months in the majority of the homes and in none was less than 4 months. Children over 3 and under 12 years of age accounted for 31 of the cases. There were 5 cases of encephalitis (encephalopathy), four of these being in children, and these were the only cases for which medical care was sought. Lead encephalopathy is the most serious of the acute manifestations of poisoning in children. Aub[3] states that the acute mental changes may develop suddenly and without warning or gradually with exaggeration of symptoms such as sudden dullness, inability to concentrate and a tendency to restlessness and irritability.

In the following case there was a gradual onset of similar symptoms in a young boy, which led his mother to call a physician who found him in a stuporous condition and promptly admitted him to hospital.

REPORT OF FATAL CASE

This patient, Benny V, a boy of 7, was admitted to the Kingston General Hospital on December 17, 1952, at 11.30 p.m. in a semi-comatose condition. Information given by his mother indicated that the boy had been delicate from birth. About two weeks previously he awakened one morning at 2 o'clock complaining of nausea and abdominal cramps but after vomiting fell asleep. Next day he was able to play with the other children. On subsequent days he had four or five attacks of nausea, abdominal cramps and vomiting all in the early morning; during the past two weeks he had become very drowsy and lethargic and had lost his appetite. Two days before admission to hospital he became so ill that he remained in bed, unable to retain any food and com-

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plaining of abdominal pain. On the morning of December 17 his mother noticed that he appeared to be delirious: his head and eyes were turned towards the right side and he frequently said that he was "going to fall".

Examination on admission. The boy was unkempt, very pale, poorly nourished and unconscious. His head and eyes were deviated toward the right: the pupils were widely dilated and reacted well to light. The teeth were carious and a bluish discoloration of the gum margins of the molar teeth was noted. The pulse was slightly irregular and averaged 75. Blood pressure 110/75. The right leg was rigid: bilateral foot and wrist drop were present.

Progress. The severe neurological involvement indicated a bad prognosis; nevertheless active treatment was commenced with intravenous fluids (calcium gluconate and physiological saline) and tube feeding. During treatment, the patient had several convulsive seizures—eyes and mouth twitching and drawn to one side, twitching of the extremities with tenseness of the body. The seizures lasted 5 to 10 minutes. The patient did not regain consciousness and succumbed on December 19, at 8.35 p.m.

An x-ray photograph of the knees was taken on December 18; the dense bands at the growing ends of the femora and tibiæ indicate lead deposition (Fig. 1).

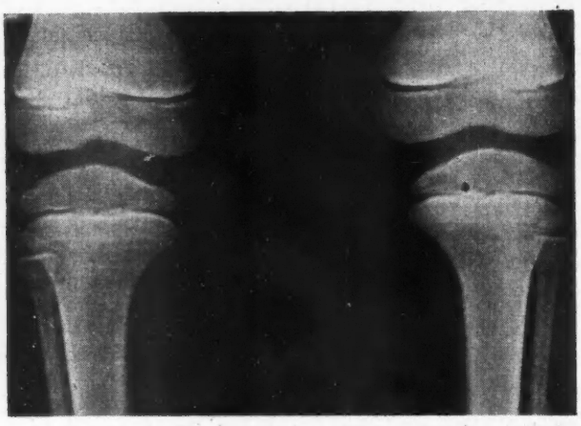

Fig. 1.—X-ray photograph of knees of B.V., showing dense bands at the growing ends of the shafts. The lines appearing beyond the bands may represent two periods of lead deposition of much less amount.

INVESTIGATION

On visiting the home in company with the physician, conditions of squalor, extreme poverty and deprivation were all too evident. The "shack" could only be described as a slum dwelling and consisted of an outer shed used as a kitchen, a dark living room and two small dingy bedrooms (Fig. 2). It served to accommodate the father and mother and four children aged 14, 11, 10 and 7 years. The sole mode of heating was an old cook stove in the kitchen with a vertical smoke stack passing to the outside. The pipes were leaky and emitted smoke into the rooms. Three of the inmates had no serious subjective symptoms but the mother and two boys, Benny aged 7 and Harry aged 11, presented signs of lead absorption and intoxication. Benny was seriously ill and could not be roused, Harry had severe abdominal cramps and constipation, but the mother, a very patient woman, made light of her symptoms although she had a typical lead line in the gums and weakness in the right grasp so that Teleky's sign was easily demonstrated. She stated that she had been in the habit of burning battery boxes throughout the winter months during the past three years as the family was on public relief and she could not afford to buy coal. She was surprised at the turn of events and did not attribute the present illnesses to the use of an abnormal type of fuel.

Pieces of broken battery casings were picked up in the shed and submitted to examination in the laboratory. A light slaty-grey deposit, present as a thin film on the interior surfaces of the sides and bottom of the casings, was scraped off and analyzed chemically. Weighed samples of this deposit, presumably lead sulphate chiefly with a little lead oxide, were converted first to carbonate and then to oxalate: the oxalate was decomposed in dilute sulphuric acid and the separated oxalic acid titrated with 0.1 N potassium permanganate. The average of three samples indicated 89.62% as lead sulphate. A portion of the casings was thoroughly washed, dried and powdered and subjected to tests for lead with negligible results.

Blood films taken from the mother and the children were stained for basophilic stippling of the red blood cells in the Industrial Hygiene Laboratory of the Ontario Department of Health using Bishop's[4] technique. The following results were obtained, the stippled cell counts being expressed as per million red blood cells (Table I).

The counts shown in Table I are very high; even if we exclude the counts of fine stippling, the presence of 22,800 medium and coarse stippled cells per million red blood cells in Benny's blood smear indicates an extremely high exposure to lead.

A 24-hour sample of urine collected from Benny between December 18 to 19 and a sample of blood and of cerebrospinal fluid withdrawn on December 19 were submitted for quantitative estimation of the lead content. A sample of Harry's urine was also obtained. Careful chemical analyses were carried out by well proved methods using dithizone for the lead extraction. The results are shown in Table II.

Fig. 2.—Back view of dwelling showing outer shed used as a kitchen and smoke pipe emerging through roof from an old cook stove—the sole source of heat.

Sitgreaves and May[5] analyzed 226 samples of pooled *normal* human blood for lead concentration by the dithizone method and obtained values ranging from 0.0151 to 0.0397 mgm. of lead per 100 grams of blood with a mean value of 0.0247 mgm. A report of the Industrial Hygiene Section of the American Public Health Association[6] states that in *normal* individuals the concentration of lead in the blood ranges from 0.01 to 0.08 mgm. per 100 gm. of whole blood and in the urine from 0.005 to 0.12 mgm. per litre in spot samples of 50 ml. or more, the mean value being 0.03 mgm. per litre.

Porphyrin determinations were also carried out on the urine samples from Benny and Harry, by the aid of a Uvispek Spectrophotometer. Coproporphyrin was estimated by the method devised by Schwartz, Zieve and Watson,[7] and uroporphyrin by the method of Sveinsson, Rimington and Barnes.[8] In experience with these methods in this department, average normal values for coproporphyrin and uroporphyrin are 14 μg. % and 5 μg. % respectively.

TABLE I.

	Type of stippling				
Name	*Fine*	*Moderately fine*	*Medium*	*Coarse*	*Sum of medium and coarse stippled counts*
Mrs. F.V.	1,900	11,200	9,000	3,700	12,700
Harry	2,400	10,000	9,400	1,600	11,000
Ben	5,500	20,000	17,000	5,800	22,800

TABLE II.

	Lead content			*Porphyrin content in urine*	
Name of patient	*Blood*	*C. S. fluid*	*Urine*	*Coproporphyrin*	*Uroporphyrin*
Benny	0.335 mgm. per 100 gm.	0.0025 mgm. per 100 ml.	0.1955 mgm. per 100 ml.	17 μg. per 100 ml.	18 μg. per 100 ml.
Harry			0.050 mgm. per 100 ml.	32 μg. per 100 ml.	7.5 μg. per 100 ml.

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SUMMARY

A family outbreak of lead poisoning is described, due to inhalation of smoky fumes arising from discarded storage battery casings in an old cook stove in a slum dwelling. Three of the inmates—the father, a boy aged 14 and a daughter aged 10, did not complain of subjective symptoms, but one boy aged 7 died from lead encephalopathy, another boy aged 11 had abdominal colic and the mother had paresis of the right wrist and a typical lead line in the gums. Although the mother stated she had carried on this practice for the previous three winters without untoward effects, it is believed that intensity rather than duration of exposure was responsible for the outcome. A slaty-gray deposit scraped from the interior of the battery casings yielded approximately 90% lead sulphate. The respiratory tract is the most important portal of entry for lead into the human body; signs of intoxication develop more quickly than when 10 times as much lead is ingested.

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RÉSUMÉ

L'auteur décrit un cas d'empoisonnement collectif des membres d'une famille, par l'inhalation de fumée provenant de l'incinération de vieux accumulateurs dans un poêle défectueux. Trois d'entre eux, le père, un fils de 14 et une fillette de 10 ans n'offraient aucun symptôme subjectif, quoique un autre fils âgé de 7 ans soit mort d'encéphalopathie saturnine, qu'un autre fils de 11 ans ait eu des coliques abdominales et que la mère ait présenté une paralysie antibrachiale droite et un liséré de Burton. Même si la mère a prétendu avoir brûlé de ces piles à plusieurs reprises au cours des trois hivers précédents, sans incidents fâcheux, il semble que se soit l'intensité plutôt que la durée de l'exposition qui ait déterminé l'empoisonnement. L'analyse d'une substance grisâtre prélevée à l'intérieur de ces piles, démontra une concentration d'environ 90% de sulphate de plomb. Les voies respiratoires sont le principal mode d'accès du plomb dans l'organisme. L'intoxication se produit ainsi plus rapidement que si le sujet faisait l'ingestion d'une quantité de plomb dix fois supérieure. M.R.D.

THE MANAGEMENT OF COLOSTOMY*

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DEVINE OF MELBOURNE once had a patient who declared that he really preferred his colostomy to a natural anus because it did not develop piles. To the average patient, however, a colostomy is a poor substitute for the customary portal of fæcal evacuation. Throughout the years many of the best surgical brains have tackled the problem of trying to build a better colostomy and the procedures which they have elaborated have been remarkable for their ingenuity if not for their functional success.

Perhaps the simplest was the skin flap devised by Payr to cover the outlet of the colostomy like a lid. It was obviously impractical and it never enjoyed any degree of popularity.

Attempts were made to place the colostomy in a position where it could be controlled by external pressure. Witzel, for example, suggested drawing the loop of sigmoid colon through the subcutaneous tissue over the crest of the ilium and out through the skin about two inches lower so that it could be compressed against the iliac bone with a bandage. He reported the case of a patient whose colostomy worked well for six years following this operation. Lenkinheld and Borchardt maintained that the bandage was superfluous and that, with this method, the new anus was absolutely continent for both fæces and gas. Roux, of Lausanne, also used bone against which to compress the bowel. In a description of the operation Mademoiselle von Mayer states that the loop of sigmoid was brought out in the midline between the intact rectus muscles in the suprapubic region and was placed in a v-shaped gutter gouged out from the symphysis pubis. The lower loop of the colostomy was kinked against the bone and thus occluded and the upper loop

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could be compressed between the bony symphysis and a supporting pad.

Bailey and Tuttle performed colostomy by bringing the sigmoid loop through a subcutaneous tunnel in the iliac fossa. Braun carried a single-barrel colostomy in a similar manner down the anterior surface of the thigh. Andrea suggested placing the subcutaneous tunnel parallel to the outer border of the rectus muscle where it could be compressed by a simple belt worn around the trunk. He felt that the angular deviation of the intestine and the diminished peristaltic movement caused by the presence of plentiful adhesions were additional advantages obtainable by this method. The operation was further modified by Burrows and Burrows who placed the bowel inside the rectus sheath rather than just under the skin.

A clever attempt at controlling the outlet of a terminal colostomy by means of a metal clip held in place in a tubular skin-graft passing below the bowel just proximal to the artificial anus was described and claimed by so many authors that it is hard to discover who actually conceived it in the first place. It was probably the brain-child of Goldschmidt who derived inspiration for the tubular graft from the various procedures he saw being used in kineplastic amputations at Sauerbruch's Clinic but the method was described with various modifications by Kurtzahn, Hans, Francois, Cuneo, Haecker, Kappis, Kleinschmidt, Ribas, del Valle, Yodice and Ciarlo. Condemning this operation as too elaborate, Lambret, in 1922, proposed the "anus en trompe" or "spout-shaped anus". This was a terminal colostomy covered by skin flaps which projected for about four inches from the abdominal wall so that it came to resemble a penis in shape and appearance. It was claimed that the anus could be rendered continent by compressing the projection against the abdominal wall with adhesive strapping. Hayem modified the skin flaps to improve their blood supply and the operation was advocated in the American literature by Briscoe.

To construct a functioning artificial sphincter has been the goal of many surgeons. As early as 1888, Maydl used the oblique muscle-split operation which was re-introduced for appendectomy six years later by McBurney, hoping that some sphincteric action might be obtained. Hartmann who used the same incision claimed that it had been originated by one of his old students, Du Bouchet. He found that the patients did have some degree of muscular control, although there was no real sphincteric action. A trans-rectus incision, modelled on the principle of the Howse gastrostomy was suggested by von Hacker in 1890 and by Weir in 1891. The principles of the Frank gastrostomy were embodied in a method described by Garrigues in 1908. The bulkiness of the spur in the double-barrel colostomies defeated any attempt at control by the muscles in which they were situated and to overcome this obstacle the terminal rectus colostomy, in which the lower loop was closed and dropped back into the abdomen, was introduced by Lane. Gersuny endeavoured to achieve sphincteric control by twisting the upper loop of the colostomy through 180 to 160 degrees in its longitudinal axis, but according to Weir, he failed to secure even moderate success. The operation was modified by Lillienthal, who first drew the terminal part of the proximal loop through the rectus muscle and then twisted it. He said that the intestinal contents became arrested behind the rectus and that, on digital examination, the opening presented two distinct sphincters, one at the site of the twist and another at the point of peritoneal fixation. He claimed that a perfect result could be anticipated within a month with absolute control of motions and enema fluids.

Under the impressive title of "sphincteropoiesis", Bernays described a plastic procedure in which the circular muscle fibres of the bowel itself were caught up in a series of longitudinally-placed catgut sutures and matted together to form a circular band resembling a sphincter. The results of this operation, according to Tuttle, were not satisfactory. Ryall endeavoured to fashion a double sphincter from fibres of the rectus muscle. Diagrams of this operation give the impression of a highly-efficient sphincter but, as pointed out by Cuneo, the resemblance can only be structural where the essential nerve supply from the autonomic nervous system is lacking. Kaiser, in 1921, was responsible for designing a terminal colostomy in which the bowel was placed under the sartorius muscle in the thigh and employed the passive tension of that muscle to control the outlet. Undoubtedly the most complicated operation of this type was proposed by Spivack in 1932 when he suggested that the ileo-cæcal valve, reversed, should be made to play the part of an internal sphincter. The operation involved division of the ileum and

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bringing out the distal end as an abdominal anus while the proximal end was inserted into the lower part of the cæcum.

Of the many attempts at the development of an artificially controlled anus, Rainey states that not one has survived its author's recommendations and Lockhart-Mummery, from personal experience of the various operations designed to increase control has found that any valvular action is soon lost and that, in fact, the results are worse than with the simpler procedures because a larger opening has had to be made in the abdominal wall in the first place. Furthermore, experience gained during the recent revival of "sphincter-saving" operations for malignant disease of the rectum has demonstrated the importance of the so-called "ampullary" reflex in determining sphincteric control. This system of afferent impulses arising in the mucosa of the lower two-thirds of the rectum signals the arrival of fæcal material in that organ and its integrity is essential to the correct co-ordination of continence or to effective evacuation, even when the sphincteric mechanism is intact. Hence it appears that any colostomy, created of necessity from a piece of bowel devoid both of the necessary reflexes and of sphincteric control, must inevitably present what Paul of Liverpool referred to as "the disadvantage of using the living intestine too much like a piece of hose-pipe". Perfect control is unattainable.

Four main problems confront the patient who has to live with a colostomy. (1) Regulation of the stools. (2) Selection of an appropriate diet, (3) Care of the surrounding skin. (4) Choice of a suitable appliance.

At first, the new anus may be a little unruly, but, in the course of a few weeks after the operation, it usually settles down to regular habits and acquires some rhythmicity of action which is at least predictable. According to Jentzer, the musculature of the colostomy hypertrophies and becomes adapted to form a sort of sphincter under the control of an ano-spinal centre in the cord. When the habit of daily evacuation has once been established, a well-formed colostomy often gives little more trouble than a normal bowel. Some patients prefer to keep the bowel empty by means of irrigations and there are many types of irrigating apparatus on the market for home use. Typical examples are those of Bowman, Nitch, Knight and Greer. Some of them are almost automatic and leave the patient with his hands free to shave, smoke or read the morning newspaper whilst enjoying his matutinal irrigation. Their chief disadvantage is that their use is time-consuming. One of Dubois' patients, for example, stated that she could not take an irrigation in less than two hours, counting the time it took to tidy up the bathroom afterwards. Occasionally this may be an advantage as there is no doubt that for some patients two hours of harmless hydrostatics would have a more salutary effect than a similar period spent in idle self-commiseration but, for the majority of cases, it is quite unnecessary.

Drugs have a place in the control of a colostomy. The most useful one is paregoric. One or two teaspoonsful may be taken at a time to check excessive flow or as a prophylactic to set the mind at rest before attending a social function. Bismuth subnitrate, an inert and harmless powder, may be used in doses of a teaspoonful or more several times a day to render the stool more solid or to deodorize it. Other deodorants are charcoal and the various chlorophyll preparations. In addition, it is often advisable to spray an aromatic germicide on the inner part of the dressing. Bulk-formers, such as methyl-cellulose and Siblin, are often useful to give the stool the correct consistency. When diarrhœa occurs, it should be remembered that it may be gastrogenous in origin, secondary to an achlorhydria, which may be checked rapidly by small doses of hydrochloric acid. Laxatives are absolutely contra-indicated. "No one" says Druckerman, "who has spent several hours with a patient suffering from diarrhœa will need to be cautioned against the use of purgatives". Where medicinal assistance is required in getting a colostomy to move, the indication can usually be met by giving mineral oil by mouth in half-ounce doses, or by an oil-retention enema instilled into the proximal loop. When a colostomy proves particularly hard to manage, the cause is usually some co-existing pathologic lesion, such as; (1) established intractable infection in the colon above the opening; (2) constipation, often in part due to the increasing necessity for morphine; (3) invasion of the colostomy by a growth spreading upward from the rectum; (4) recurring prolapse; or (5) intractable diarrhœa associated with intra-abdominal metastases.

An elaborate diet sheet is not often needed by the patient with a colostomy. Experience will

soon tell him what food he can eat and what will cause him distress. As a general rule, the diet should be dry and constipating, fats should be limited and vegetables and fruits liable to cause excess peristalsis should be avoided, but often patients can eat these substances without inconvenience. Many patients find that their colostomies are not disordered by a moderate intake of beer or liquor. To place unnecessary restraint on the patient's diet brings hardship not only to him but also to the housekeeper who has the trouble of preparing extra dishes for him. Eating should be encouraged rather than restricted but where it is found necessary to adhere to a strict constipating diet, care should be taken to ensure that the patient receives an adequate supply of vitamins, especially of vitamin D.

The skin around a colostomy requires special care. Although dermatitis may arise through irritation by small amounts of trypsin in the discharged fæces, in most cases, it is the result of lack of personal cleanliness. Dressings should be changed as soon as they are soiled and the colostomy and surrounding skin should be cleansed with soap and water. When the irritation is severe, it can be alleviated by protecting the surrounding skin from fæcal discharge by smearing it with zinc oxide or aluminum paste. Patients sometimes complain of bleeding from a colostomy. When this occurs, the site will usually be found in granulation tissue between the bowel and the skin-edge and the bleeding can easily be checked by the use of silver nitrate.

The colostomy belt should be the simplest possible. According to Cattell, 70% of patients can manage their colostomies without an appliance at all. A well-fitting abdominal belt is generally all that is required and often this can be made by the patient himself. Where there is any tendency to prolapse, the belt may be reinforced with a flat celluloid or plastic plate to cover the site of the colostomy opening. On no account should a cup be worn as it may cause prolapse or herniation and particularly undesirable are rubber bags which have the additional disadvantages of absorbing and retaining fæcal odours. Disposable soft plastic bags are now available and these may be useful in emergencies or when there is nocturnal incontinence. The main bulk of the dressing should be of cellu-cotton which can be disposed of in the toilet. A layer of gauze or wool may be placed over this to prevent it from fraying but it should be remembered that these substances are insoluble in water and must be burnt, which makes them inconvenient especially when travelling. For those who dislike passing flatus audibly, Hirschman recommends placing a pad of cotton directly over the opening to act as a muffler.

Certainly colostomy is not a desirable thing and yet there are patients in every walk of life who live happily in spite of having a colostomy, dancing, playing golf and tennis and even indulging in heavy manual labour. Much depends upon the patient's mental attitude towards his affliction. Hullsiek expressed the belief that eight out of ten doctors would refuse to submit to colostomy if they were unfortunate enough to have rectal cancer. But there is still much wisdom in the philosophy of Mr. Daniel Pring of Bath, one of the first English surgeons to perform the operation at the beginning of last century. "I am of the opinion, on the whole," said Mr. Pring, "that an anus in the side is better than no anus at all".

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FATAL DUODENAL HÆMORRHAGE IN THE NEWBORN*

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OF ACUTE PEPTIC ULCERATION in the newborn, Moynihan[1] wrote "In these cases the onset of symptoms is sudden, their development rapid and the end is swift". Most observers both before and since, including ourselves, have been forced to this same unhappy conclusion. The obstetrician may well be the first to encounter this emergency in the newborn. Because its prompt recognition is imperative if anything useful is to be done, we wish to re-emphasize the real existence of this entity in the newborn and to review its clinical aspects.

The literature is replete with collected accounts and statistical analyses of peptic ulceration in adults, but those in childhood and infancy number relatively few. Holt[2] compiled a series of 95 cases of duodenal ulcer in infants under one year of age of which 9 manifested symptoms in the newborn period. Helmholz[3] collected 91 cases of peptic ulceration occurring in early infancy. Of Theile's[4] review of 248 cases of peptic ulcer in the young, 119 occurred during the first month of life. Bird, Limper and Mayer's[5] study of 243 cases included 38 occurring during the first two weeks of life and another 51 before the end of the first year. Only one of Guthrie's[6] 9 cases died during the newborn period, but the first four months accounted for 7 of the remainder. Lee and Wells[7] in reporting their case advanced evidence pointing towards perforation of a gastric ulcer actually *in utero*. The findings of these authors and others[8 to 16] indicate that if peptic ulceration in infancy is uncommon, its recognition is even rarer. The number of times the diagnosis is first made at the autopsy table would lend weight to this impression.[17] As Palmer[10] suggests, it is perhaps more unlooked-for than uncommon.

The mother in this case was a healthy primigravida, aged 23 years, and the pregnancy had gone to term. The birth was spontaneous after an episiotomy had been performed. The baby, 3,005 grams, breathed and cried lustily at birth. Her condition was perfectly satisfactory for the first two and a half days. One evening, 63 hours after birth, the baby was found in her cot to be white and cold. A dark, bulky stool was passed at this time and was quickly succeeded by two smaller bloody motions. The hæmoglobin was 13.4 gm. %. Vitamin K was given intramuscularly.

The baby's condition rapidly deteriorated. Her pulse became barely perceptible at the wrist and the rate uncountable over the heart. A dark trickle of blood appeared at the corner of the mouth. Laryngoscopy revealed a clear airway but the presence of bloody mucus in the pharynx. A gastric tube was passed and approximately 5 ml. of altered blood were aspirated from the stomach. The foot of the cot was elevated, continuous oxygen was administered and 80 ml. of compatible blood was infused into the left femoral vein at the level of Scarpa's triangle. The infant improved only temporarily with these measures; soon there was a reversion to the former marble-like pallor followed by the onset of Cheyne-Stokes breathing. Death occurred three hours after the baby's appearance had first given cause for concern.

Autopsy was performed one and one-half hours after death. The gastro-intestinal tract, from the stomach to the rectum, was filled with jelled blood mixed with meconium. On the posterior wall of the first portion of the duodenum was found an area of acute ulceration 3 mm. in diameter. No other site of intestinal hæmorrhage was demonstrated. On microscopic examination the ulcer was seen to affect the mucosal and submucosal coats and its base was covered with fibrin, necrotic debris and leukocytes which extended into the muscular layers. All the coats in the neighbouring walls were infiltrated with neutrophils.

The only other abnormality discovered by the pathologist, apart from the appearance of exsanguination, was a slight thickening of the walls of the alveoli of the lungs indicating a moderate degree of congenital alveolar dysplasia. Permission for examination of the brain was not granted.

ETIOLOGY

Infection.—Several authors[1, 6, 25 to 28] report the occurrence of gastro-duodenal ulcers along with acute septic infections such as meningitis, pneumonia, septicæmia and acute appendicitis in older children and adults. While they do occur in the newborn[29] such co-existing lesions must be uncommon in this age group. Dunham and Shelton[24] have described one case of gastric ulceration following staphylococcal omphalitis and septicæmia. Helmholz[3] was able to demonstrate diplococci and streptococci in nearly all of the 24 specimens of gastro-duodenal ulceration he examined. Mills[11] described a fatal case of gastric ulcer in a six day old infant and sections at autopsy revealed streptococci in the tissue about the ulcer. Thelander and Mathes[31] also postulated neonatal sepsis as the causation in their case and pointed out that in the newborn the classical signs of infection are often lacking. This peculiar want of response to bacterial invasion may, therefore, result in failure

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to ascribe the proper cause to some cases of neonatal peptic ulceration.

Circulatory causes. – Landau[21] writing on melæna in the newborn in 1874 suggested that thrombosis of the umbilical vein and subsequent embolization might cause occlusion of a duodenal blood vessel accompanied by peptic ulceration of the overlying mucosa. Brockington and Lightwood[13] point out that evidence of infarctive phenomena elsewhere is lacking in these cases.

That a vascular disorder may have some relationship to the typical duodenal ulcer on the posterior wall above the ampulla of Vater and may have some etiological significance is strongly suggested by the well-known finding of Wilkie of Edinburgh.[22] He demonstrated that the supraduodenal artery, the vessel supplying just this area, has very poor anastomotic connections.

In suggesting that an extreme degree of congestion of the abdominal vessels during the birth process may in some way cause ulceration Shore[23] postulated a sequence of congestion, rupture and interstitial hæmorrhage from the gastric and duodenal vessels. Guthrie[6] felt that asphyxia with consequent duodenal congestion and mucosal hæmorrhage or ischæmia resulting from vascular spasm might be a responsible factor. In addition to these possible circulatory accidents, the peculiar properties of the gastric juice in early infancy may play a part. Miller[34] has shown that following birth gastric acidity rises to a maximum level within 24 hours postpartum after which it drops off considerably.

Neurogenic causes.—It is well known that gastro-duodenal ulceration may be present with various cerebral lesions. Rokitansky[35] over a century ago suggested cerebral disease, causing disturbances in the innervation of the stomach, may produce "gelatinous softening" of that organ associated with extreme acidification of the gastric juice. Cushing[36] drew attention to the probable rôle of intracranial hæmorrhage of the newborn upon subsequent peptic ulcer formation and he cited as illustrations Elsaesser's series of no less than 38 cases of "autodigestion" of the stomach, in the newborn infants, many of them associated with brain damage. Other workers[37 to 42] have concurred with Cushing that the dual lesion of brain (hypothalamic) injury and gastro-duodenal ulceration is not merely fortuitous.

Cerebral hæmorrhage has occasionally been found to be associated with gastro-duodenal ulceration.[32, 43, 44] Cerebral anoxia and œdema in the newborn have been occasionally found to be associated with peptic ulceration.[44, 45] Mossberger[46] reported the occurrence of a perforated duodenal ulcer in a five day old infant co-existing with a tumour in the region of the hypothalamus.

Stress.—In line with Selye's reasoning in his explanation of the general adaptation syndrome it might be suggested that labour, especially if long drawn out and difficult, may comprise the non-specific stress which could induce in the fetus and newborn infant the alarm reaction to such stress with its concomitant sequence of hyperadrenalism and alterations in gastric juice composition. With the super-imposition of additional accidents such as hypothalamic injury due to hæmorrhage or anoxia, sepsis or trauma to the gastro-duodenal mucosa or mechanical interference with its blood supply, acute erosion or ulceration could follow. At the birth of the infant and the cessation of the stress, healing of the ulcer would be the rule providing the complicating injuries were not soo severe and hæmorrhage or perforation did not ensue.

Pathology.—In peptic ulceration in the newborn, males outnumber females very slightly.[1, 2, 5] Duodenal ulcers are about twice as common as gastric ulcers and are generally situated on the posterior duodenal wall[6, 10, 22] and almost invariably above the level of the ampulla of Vater.[5, 17] Gastric ulcers are located, in order of frequency, near the pyloric ring, on the posterior wall, in the cardia or on the anterior wall.[17] Ulcers occurring elsewhere such as in the œsophagus or Meckel's diverticulum have also been recorded but are rare.[17] Ulcers may be single or multiple.[6, 10] They may vary in extent from a pin-head-sized ulceration of the mucosa to a 3 cm. perforation of the stomach.[10, 43] Schlumberger refers to large, poorly demarcated, shallow areas of gastric mucosal dissolution as gastro-malacia.[44] These are to be differentiated from the small, relatively punched out, sharply circumscribed ulcers. They appear to be quite definitely of ante mortem origin.[35, 43, 44]

Typically, peptic ulcer of the newborn has an acute, punched-out appearance;[5, 14, 47] less commonly it has sloping, terraced or even undermined borders.[22] One or more eroded or thrombotic[9, 13, 17, 22] vessels are sometimes seen in the ulcer's base. Clots are often found in the stomach and bowel and sometimes filling the ulcer crater,

leaving no doubt as to the source of the hæmorrhage.[6, 10, 13, 15, 44] Often, perforation occurs through all the coats of the stomach or duodenum. On occasion, pancreas or omentum has plugged a potential perforation.[6, 44]

The third common complication of peptic ulcer in adults, namely cicatricial pyloric stenosis, does not occur in the newborn although co-existent congenital hypertrophic pyloric stenosis has been reported.[32, 48, 49] Pylorospasm associated with duodenal ulcer occurs in the newborn while organic pyloric obstruction does not infrequently complicate the more chronic ulcers of older infants and young children.[5, 10]

Histologically, the usual finding is a striking absence of fibrosis or fibroblastic proliferation about the ulcer cavity, the lesion being chiefly destructive with little attempt at repair.[2, 3, 6, 24, 31] This finding is interpreted as evidence of extreme rapidity of development and progression of the ulcer to a fatal termination before there is time for the occurrence of any noteworthy cellular reaction. A few observers, however, report not only an extensive polymorphonuclear reaction about the ulcer but bacteria in the Gram's stained sections of it. Helmholz and Gerdine, like Rosenow,[30] were able to isolate in pure culture S. *viridans* from the peptic ulcers of infants.

Not all neonatal peptic ulcers progress to a fatal termination without therapy,[6, 8, 12, 33, 50, 51, 52] some undoubtedly healing rapidly and spontaneously. Well healed scars have been found and in one instance the ulcer was quite well healed after a severe hæmorrhage.[11] As before mentioned the various intracranial lesions sometimes found to accompany the ulcer are hæmorrhage, tumour and the histologically demonstrable effects of anoxia upon the brain cells.

CLINICAL ASPECTS

Usually there is little at birth that would give any indication of subsequent events, the infant appearing healthy, well developed and breathing and crying spontaneously. Occasionally, following cerebral anoxia or hæmorrhage, the baby is born shocked and limp and later signs of gastro-intestinal bleeding or perforation are superimposed.[32, 44] Usually, however, no such symptoms precede the sudden and catastrophic onset of melæna, hæmatemesis or peritonitis following perforation. Less common signs are diarrhœa, vomiting, failure to take feedings well, dehydration and pain as manifested by screaming and drawing up of the knees.[14, 18, 47, 51]

The occurrence of bleeding is usually completely unexpected, commonly occurring first between the 24th and 96th postnatal hours. The usual signs characteristic of severe blood loss will be present; pallor, restlessness, tachycardia and tachypnœa, hypothermia, clamminess of the skin, especially of the extremities, and oliguria. The napkin may be found to contain a bulky black stool and occasionally dark red blood. Hæmatemesis may occur but is usually not massive in amount.

Absence of a suggestive history of maternal immunization and a negative Coombs' test will immediately eliminate the possibility of erythroblastosis. The clotting time will be normal unless, of course, there is an associated hæmorrhagic disease. A baby will not show the clinical signs and symptoms of loss of blood if the blood in the stool or the vomitus has come from the mother's nipple.

In duodenal ulcer, associated with a marked degree of pylorospasm, vomiting, often projectile in nature, is the outstanding feature of the disease. The time of its occurrence with respect to the infant's age and the presence of blood in the stool or vomit will help to differentiate it from congenital hypertrophic pyloric stenosis.

DISCUSSION

In the case reported the cause of the duodenal ulceration was not ascertained. Perhaps the examination of the brain might have revealed a suggestive lesion but clinically, from the uncomplicated labour to the healthy appearance of the newborn baby, there was nothing from first to last to give any anxiety. Neither the mother nor the baby suffered from infection. There was no obvious neurogenic lesion or reason to believe there might have been cerebral damage and the labour and delivery being without obvious stress the so-called alarm reaction cannot have been aroused beyond what may be natural after any labour. The baby, neither at birth nor thereafter exhibited any signs of asphyxia.

It is easy to be wise after the event. The fact is, gastro-duodenal ulceration with uncontrolled hæmorrhage was not taken into consideration when only 80 ml. of blood were given intravenously. Now that we are aware of this definite possibility and of an equally likely complication of peptic ulcer in the newborn, namely, perfora-

tion, perhaps the fatal outcome of this case will not be repeated. However, if the literature on this subject is examined no single case of severe hæmorrhage from peptic ulcer successfully treated surgically or by repeated and relatively massive transfusion of blood at this age will be found up to the beginning of this year. There are, as has been indicated in the references, many cases reported where the ulceration has only been diagnosed at autopsy.

It is perhaps outside the purview of the obstetrician to lay down the possible lines of treatment but at least he should be permitted to consider them in the baby, in which he is so intimately interested.

Ladd and Gross[33] emphasize the gravity of the outlook in the case of the infantile bleeding ulcer and urge that blood be given early and in liberal amounts. The actual volume would best be based on the clinical reassessment of the infant's condition from hour to hour rather than on any arbitrary standard unless a "hot" laboratory is nearby. It is clearly impossible to gauge the volume of the blood lost into the intestinal lumen. Treatment of the bleeding ulcer continuing to bleed in the newborn is an enigma because we know of none that has been saved by any method. Suffice it to say that shock, gross melæna and hæmatemesis means the baby's life is in dire peril and will probably not be saved even by continuous blood transfusion alone. A Levine tube might be placed *in situ* in the stomach to allow a continuous drip of breast milk or diluted cow's milk combined with a non-absorbable anti-acid to counteract the acid. The course of the bleeding might be followed by aspirating some of the stomach contents which will always give an indication of what is happening in the duodenum. This conservative treatment may add to the amazing recuperative powers of the infant and stem the tide of the lethal loss. A combination of transfusion, fluid replacement and surgery might conceivably avert death. In the last extremity of exsanguination the baby would probably not stand roentgen screening but in the less urgent case lipiodol might be used to show up the exact site of the lesion or the signs of perforation.

SUMMARY

1. A case of duodenal ulcer in the newborn complicated by fatal hæmorrhage has been described.

2. From a review of the literature certain aspects of the etiology, pathology, clinical features and diagnosis have been discussed.

3. Certain points are emphasized: (a) Bleeding from the gastro-intestinal tract in the newborn should not be assumed to be due to hypoprothrombinæmia until an increased prothrombin time is demonstrated. (b) A significant proportion of cases of melæna neonatorum is due to peptic ulceration. (c) The infant whose ulcer is complicated by severe and continuing hæmorrhage will probably die if concentrated conservative therapy is not applied with the closest attention to the best known techniques. Rapid surgical repair is not contemplated with reasonable optimism. (d) The place of obstetrical complications in the possible etiology of peptic ulcer has been pointed out.

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THE CANCER CLINIC*

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THE ONTARIO CANCER FOUNDATION, Kingston Clinic, was developed in 1947 as the first of its kind in Ontario, under the auspices of the Hon. Percy Vivian, then Minister of Health for Ontario. He visualized a Cancer Clinic in which all branches of the disease could be dealt with by any method of treatment and by those most competent to deal with all types of the illness. There was no difficulty in choosing a staff for this clinic, as the Medical Faculty of Queen's University, with the heads of the various services, were available and were willing to serve. The actual mechanics of appointment are simple. The University names the staff and the Foundation approves.

The reasons for setting up such a complete clinic may be considered under the following headings:

First, assistance to the patient.—Cancer in many of its phases is a most difficult disease to diagnose and to treat and it is, therefore, essential that the most highly trained personnel should be available to deal with it. Further, one type of the disease may require the services of two or more physicians, as, for example, carcinoma of the cervix, in the treatment of which the gynæcologist, urologist and radiotherapist are all required. In stating this, one should not lose sight of the fact that the general practitioner is an important cog in the wheel of control. He must be the one who first sees the patient and directs him to the Cancer Clinic. He is constantly kept informed of the progress of the disease and the treatment that is being given and in many instances he carries on treatment after the patient has left the clinic.

Second, equipment.—Radiotherapy machines are very expensive and it would be impossible for most areas to have the advanced type of equipment required to do the best work without added financial assistance. Not only is radiotherapy equipment provided, but also special diagnostic instruments, which often are too expensive to be provided by the fees received from the patients. Funds for this work are supplied by voluntary subscription and through government grants. In our own clinic it is found that the fees collected pay for less than one-half of the total cost of operation.

Third, education.—The staff have available to them publications from all parts of the world and audiovisual material which may be used to teach interns, nurses, and lay and professional groups. The Centre also serves as a source of information to physicians practicing in the area.

Fourth, the registry, which is the centre of clinical research. Some indication of this work is shown in the six-year period of operation from 1947 to 1952, when a total of 2,543 new cases of cancer were treated. Of these, the number lost sight of was 5%. In particular examples, such as the breast and cervix, this figure was reduced to between 2 and 3%. The highest percentage lost sight of was in cancer of the skin, the reason being that the patients do very well, with an overall cure rate of 99% and feel it is unnecessary to keep in touch with the clinic staff. The total number of follow-up examinations for these years was 7,889. Much credit is due to the Ontario Department of Health, and to Dr. A. H. Sellers, medical statistician of that Department, who has assisted greatly in the statistical survey.

Now, to turn to some details of operation of the clinic. In actual practice, our clinic operates as three activities combined in one, namely, the registry, the radiotherapy department and consultation and follow-up examinations. The registry covers all cases of cancer in the hospitals of this district, whether treated in the cancer clinic or not. The staffs of our two general hos-

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pitals have agreed that this shall be done. In the radiotherapy clinic, all types of cases, both malignant and non-malignant which require treatment by irradiation, that is, by radium and x-ray, are dealt with. Consultation and follow-up examinations are made by any member of the staff, including radiotherapists, surgeons, internists, urologists, gynæcologists, etc. Any of these may use the examining rooms of the clinic and may use the services of the secretarial staff to make records, communicate with doctors, register information and assess results of treatment.

Other activities of the clinic include meetings of the staff at intervals to discuss cases treated and to exchange information, and the fostering of the local branch of the Canadian Cancer Society.

To avoid misunderstanding it may be stated that no member of the clinic staff gets any remuneration directly from the Cancer Foundation except the director, who receives a salary for directing the organization. Physicians of the staff are entitled to collect fees, either directly or through the financial office of the clinic, from patients who receive their services. The patient who is unable to meet these charges pays nothing. Those who are able to pay a percentage are expected to do so. Indigent patients who require radiological examinations have these provided free by an arrangement with the Department of Radiology of the hospital by which the Foundation pays 50% of the fee.

Many in private practice fear that much of medical treatment is being taken over by social agencies and certainly it is true that such has been the case in venereal disease, tuberculosis and poliomyelitis. The reason is not difficult to find. In the first two named there is a threat to the health of the whole population and in all the financial burden involved soon becomes too great for the individual and assistance must be provided. Such a system carried to its ultimate would, of course, lead to state medicine. It is not necessary at this point to enter into a discussion of the numerous abuses associated with the latter. The point I would make is that a clinic such as this helps to solve many of those very problems which emerge in our present system of free enterprise and which, under ordinary circumstances, lead to discontent among our people. We continue to look upon the general practitioner as the first line of defence in all diseases but when the burden of care becomes too great the Cancer Foundation stands ready to take it over and to carry on with all the resources at its command.

In closing I would thank the Ontario Cancer Treatment and Research Foundation for its constant efforts to improve the operation of its clinics and to provide the best care for those suffering from cancer.

SOME PHASES OF CHORIO-CARCINOMA*

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CHORIO-CARCINOMA has always been of a mild interest to the gynæcologist: but probably because of its comparative rarity, it has been more academic than clinical and has usually been put at the end of a list of differential diagnoses. This very malignant entity presents a great many interesting and intriguing phases, all of which would bear further study.

In making a survey of the English literature of the past ten years one is impressed by certain facts: (1) The comparative rarity. (2) The high mortality. (3) The difficulty in diagnosis. (4) The incidence of late fatal metastases after apparent cure.

The literature has been very inadequate in positive information and has consisted mainly of papers like this one, namely a hasty review of the subject and reporting of a new case. One point, however, that is stressed is that a proper appreciation of the true pathological entity is essential. In studying the statistics of the Ottawa Civic Hospital, it was found that from 1930 to 1952 inclusive, there were 16 cases, 10 of which were on my service. In all these cases the diagnosis had been made preoperatively and the findings confirmed by the pathologist.

Historically Gregorini noted and reported this condition in 1795; Sanger in 1888 and Marchand first described the true histogenesis. Since then,

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up until recently 2,000 cases have been reported, but there must be a vast number of cases never reported, as, for example, our 16 cases in the Ottawa Civic Hospital.

The following questions regarding chorio-carcinoma may be asked: (1) How common is it? (2) How to make an early diagnosis. (3) The best way of treating the condition. (4) How malignant is it? (5) Is it an urgent disease? (6) What is the pathology? (7) Is it easy for a pathologist to be certain of the diagnosis? (8) What is the value of a pregnancy test and is it infallible? (9) Is it always a gynæcological disease? (10) Does it occur at any time not associated with pregnancy? (11) Prognosis.

What is chorio-carcinoma? The most simple and easiest classification to remember is: (1) *Syncytioma or syncytial endometritis.*—Hertig casts a doubt as to whether this group should be considered as a true chorionic malignancy, although most authorities put it in a malignant or potential malignant group and it should be adequately treated. These are the cases in which so many cures are reported.

(2) *Chorioadenoma destruens.*—This group of chorionic malignancy is also known as invasive or malignant mole. The prognosis is usually favourable as metastases are not common; the fatal cases are usually due to perforations or hæmorrhage.

(3) *Chorio-carcinoma.*—The cases in this group are very malignant, metastases free and wide and the mortality rate is about 100%.

I have, as you see, simply stated the classification and do not intend to enter into a long pathological explanation, but I do want the importance of cellular pathology strongly stressed. One must have a comprehensive knowledge of the subject before being able to adequately and satisfactorily treat a patient. This point once again demonstrates how extremely important the knowledge of pathology is to the clinician and how dependent we are on the pathologist. I would stress the fact that you cannot and should not expect the pathologist to make a positive diagnosis on a few curettings; sometimes even with the whole uterus available it may be difficult.

How common is it? Is it such a rare disease that it is only of academic interest and put down in the differential diagnosis as an after-thought, or should we always keep it in mind? Without quoting a great many figures, I would like to state that roughly chorio-carcinoma has an incidence of one in about 14,000 pregnancies, hydatidiform mole one in 2,500 pregnancies and 1% of moles become malignant. These are approximate figures and seem to vary with the authorities and depending on histological criteria used. A very interesting and illuminating paper by Acosta-Sison from the Phillipines reports from 1941 to 1949 122 cases, all proved by their pathological department. As you see, this is a phenomenal number; no clear explanation given, but attention drawn to the large number of quick pregnancies and nutritional deficiencies. India also reported a very high incidence more or less under the same condition and economic status. It gives one food for thought, also one wonders if their pathological criteria are of the same standard as we follow.

Acosta-Sison states that chorio-carcinoma is any pathological invasive growth of the chorionic epithelial cells. All other classification is purely of microscopic interest, but from a purely clinical point of view all varieties are malignant and may cause death in some way or other unless adequately treated. Even though it is a rare disease, as you see, I definitely think that it is more than an academic one. It must be remembered that every pregnancy, either normal intrauterine, ectopic, or an abortion is a potential chorio-carcinoma. It may and does occur. In any case of pregnancy that has any bleeding not absolutely proved as to cause, it must be ruled out. The malignant process may start very soon after fertilization, and metastatic lesions have been proved to be present even before the responsible pregnancy has been expelled.

The following case is of interest from many angles.

A young woman was admitted to the medical side suffering from an apparent severe cerebral accident which proved fatal: gynæcological examination was negative and there was no recent vaginal bleeding. However, it was found out that following her normal delivery nine months before, she had had some post-partum bleeding which persisted for some time but cleared up without surgical treatment. Autopsy showed a large metastatic brain lesion of chorio-carcinoma.

How to diagnose the disease or I should say, when to suspect the entity. To repeat, it should be suspected in any case associated with any form of pregnancy that has any bleeding of which the exact cause is not proved. This is a trite aphorism but a true one. Unfortunately, there are so many variations of bleeding that

the proof is indeed difficult. Pregnancy tests, as you can readily see, are of no great value. A positive one just proves a pregnancy already recognized, a negative report gives a sense of false security. However, as most cases occur after the expulsion of the pregnancy, clinical acumen is then of great value. In any case of bleeding after complete expulsion of the pregnancy, ask the following questions: (1) *Does the history fit the case?* Pregnancy is known, the products of conception are expelled: why bleeding? (2) *Is the uterus enlarged?* Why? The feel of the uterus —is it soft? Why?

If these questions cannot be satisfactorily answered, then one is justified in suspecting the disease.

What is the status of a pregnancy test and is it infallible? In a situation as stated above, one naturally wants a test. A positive one, as I said before, does not answer the question, are you prepared to do major surgery in a young woman? If negative, are you still satisfied that all is well? Remember, you are dealing with a possibly highly potential malignancy and undue delay is not wise. I would repeat the pregnancy test and watch the blood picture. A dilatation and curettage is indicated but, as I have already stated, may not be positive but may give a lead. If you are sure of the accuracy of your history and pelvic examination then I would advocate surgery. In a very recent case we had such a history of negative pregnancy test. Operation was delayed, symptoms persisted, again a negative test, operation was performed and chorio-carcinoma was found.

The literature is full of similar reports, both in early and late cases, and in numerous case histories of a negative pregnancy test just before a fatal metastatic lesion. In the Scandinavian literature there is an excellent article on the value of frequent quantitative tests for chorionic gonadotrophic hormones. All this does not mean that we are to ignore pregnancy tests. Far from it, but I do say we are to evaluate them in accordance with symptoms and physical findings.

How malignant is this condition and are these cases urgent? There is a belief that all cases of chorio-carcinoma are fatal, yet the literature does report "cured" in many authentic cases. Many live for years, 8 to 10, then die with brain or lung metastases. The question resolves itself into a careful pathological study. This is an urgent disease. One never knows when secondary malignant growth may occur and there is always a danger of uterine perforation and fatal hæmorrhage.

Treatment.—This is the easiest question to answer. Operation. Clean out the pelvis and remember metastatic lesions are not a contraindication for surgery because it is a well established fact that often these lesions disappear after the primary tumour has been removed, whether or not permanently, I cannot say. Even in some of the advanced cases I think operation is justified. It may prolong life and will certainly prevent fatal hæmorrhage or peritonitis. Roentgen-ray is of doubtful value, both as regards local and metastatic lesions, although it has been tried as one always does.

Prognosis.—Again depends on the accuracy of the pathologist. Pregnancy tests and x-ray studies are of great value in the follow-up.

To quote one case.

A young married girl, age 23, past history and family history essentially negative; in fact, an excellent family history of health and healthy pregnancies. Husband's family history—a very healthy one. Pregnancy occurred very soon after marriage. Her family physician, when first seeing her, thought that her uterus was larger than indicated. A few weeks later there was some very brisk vaginal hæmorrhage. Bed-rest with cessation of bleeding. Two weeks later severe hæmorrhage with passing of a couple of buckets of what was apparently typical hydatidiform mole. Uterus appeared to contract and return to normal size. A week later, slight vaginal bleeding, severe abdominal pain with shock and uterine enlargement.

She was admitted to hospital with an admission diagnosis of possible chorio-carcinoma. She appeared to be in fairly good health. Vaginal examination showed a slightly enlarged soft uterus, a palpable ovary, no adnexal masses. The usual laboratory studies were done. Pregnancy test faintly positive. This meant nothing in view of the history. Kept in hospital for about 10 days. Looked and felt well. No bleeding. Pregnancy test now negative. Diagnostic D. & C. told us nothing. Discharged: to report in two weeks. Pregnancy test again negative. Examination: Uterus firm. Again sent home. Returned in a month. Some slight bleeding. Enlarged uterus. Was it a normal period? Pregnancy was possibility. Pregnancy test positive. Admitted to hospital. A diagnostic D. & C. done which was not very informative and operation advised. Pathological report—syncytioma with some invasion.

This case was largely determined by history. Was it handled correctly? If we had not operated, would the condition have progressed rapidly to a fatal termination or would it have righted itself? I feel that I would act in the same way if the case arose again.

CONCLUSION

This whole subject is indeed an important and complicated affair with many repercussions. I have only touched on a few obvious points.

There are a great many phases that should be more fully discussed.

Although it is a rare disease it does occur; it must be handled carefully, quickly, and with close co-operation of the pathologist if one is to obtain good results.

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THE TEACHING OF UNDERGRADUATE RADIOLOGY AT QUEEN'S*

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"Come, come and sit you down; you shall not budge;
You go not till I set you up a glass
Where you may see the inmost part of you."

Hamlet, Act III, Scene IV.

AT QUEEN'S we devote a good deal of attention to the study of radiology. Undergraduates are taught the basic principles of this subject in the second year and again in the fourth and fifth years. In the sixth year they learn something of its practice. In the second year of medicine, x-ray physics is well and thoroughly presented by the Department of Physics. In the fourth year, eighteen hours are given over to lectures and demonstrations in diagnostic radiology. In the fifth year, eighteen hours are used for the teaching of therapeutic radiology and the radiological diagnosis of malignant disease. A further forty-two hours is given over to lectures and demonstrations in diagnostic radiology.

In the sixth year each student spends a week as a "shadow" in the division of Diagnostic Radiology and in the division of Therapeutic Radiology and in the Cancer Clinic. As a "shadow" he follows the radiologist during his daily routine. He sees fluoroscopy. He sits in on the routine interpretation of films. He is shown records and photographs of cancer patients. He sees patients and finds out how they are handled in both diagnosis and therapy. He watches the radiotherapy of cancer and of benign conditions. In addition to this, each student in turn presents the clinical aspect and/or the radiological side of interesting cases, weekly, before a conference group of students.

The student is not taught radiology from the standpoint of the specialist. He is drilled over and over again in the recognition of the standard radiological findings in health and disease. He is encouraged to study medicine and surgery with radiology as a help. Diagnostic radiology is described as being one method of inspection. It is one way in which a picture of the diseased process shows. It is often a very important diagnostic feature. Occasionally it is the only diagnostic feature. It may be the least important part of the clinical picture.

We feel that when our students graduate they have a good grasp of the elementary points of diagnostic and therapeutic radiology. Our graduates, therefore, should know enough about radiology that they can use it usefully and assess radiological reports at their proper value. They should also have a picture of disease which

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complements other concepts acquired in the study of other branches of medicine.

We realize that there is a danger of over-simplification of radiology, by drilling students again and again in basic principles and standard aspects of the subject. To some students this has a tendency to make the whole affair seem too easy. However, we are secure in the thought that during the final year the student must serve as a "shadow" for a week in the department. During this week he gets, or should get, a true perspective. If perchance, he knows it all, this experience soon disillusions him. He finds that radiology is filled with many diagnostic problems, and is just as complicated as other fields of medicine. The experience is of course not always salutary and once in a while there returns, for further graduate medical training, one who after a short year or so elsewhere, undertakes to instruct us. This we like to think is the result of outside contamination!

In the first clinical year the student enters upon the study of clinical radiology. During the first lecture the following points are made in something like this manner:

"You are entering upon the study of clinical radiology. It is not the intention to make radiologists out of you. You will be taught elementary principles and drilled in standard radiological findings and procedures. All this is done in the hope that radiology will aid you in the general study of medicine and surgery. You have already been introduced to the physical principles underlying x-ray machines and the production of x-rays. You will now undertake the study of the practical application of these principles, with relation to both healthy and sick people.

"When you visit a strange city you start out from a square or a circle or a hotel to explore it. From there you explore streets and other squares, enlarging gradually your knowledge of the whole. In these lectures we will introduce you to certain standard x-ray films, which will be your starting point. First you will learn about your immediate neighbourhood. Then, as you learn the near and familiar things, we will start to explore.

"When you first look at a film you observe its shadows. Do this in some sort of systematic way. Look at a chest film. You look first at the soft tissues surrounding the thorax. Next, inspect the bony parts. Next, look over the heart and mediastinum, and the domes of the diaphragm. Then, observe the lung roots, and the lung fields from above downward."

"If you find something unusual, reflect—Is this normal? Is it an artefact? Is it a congenital anomaly? Is it pathological? If so, what is it?"

Later we teach—"Think of the history and signs and symptoms. Does your idea of a radiological diagnosis make sense, when viewed in the light of the clinical findings? If not—why?

"Try to learn what you can about 'normal findings'. This will be quite difficult. In ordinary practice, unless radiology is studied particularly you may never be quite sure of what constitutes the normal. Leave that to the radiologist.

"Above all, do not try to read clinical signs and symptoms, and your working diagnosis into the film. This is a common clinical error. It is true that in retrospect you will sometimes be able to say 'Now, that possibly is early evidence of the lesion'.

"Avoid the pitfall of labelling curious markings that are within average normal limits as evidence of disease. Once, in so many times, you may be right. The other, very many times, you will be wrong and you who will have caused needless anxiety and expense to trusting patients. You must accept the fact that calculated risk of error must be taken. You must learn to deal with average normal findings. Give the radiologist the clinical facts and leave the decision regarding the reading of the film to the radiologist. The clinical decision of course is a different story. The clinical findings may completely over-ride the radiological picture. Don't however let your judgment be biased by your reading of the film."

We try to teach our students enough radiology to enable them to make a tentative diagnosis on ordinary films while waiting for a radiological report, when a radiologist is not readily available. We are very strongly opposed to the teaching that suggests to the student that, as a doctor, he should make his own report upon films. We de encourage consultation with the radiologist. We desire this in all cases, but particularly if the radiological report does not appear to measure up to the clinical findings. Teamwork is recommended.

We explain to students that the standards and elementary things we have taught them are not enough. Experience gained in seeing many, many films is necessary to arrive at proper conclusions and develop good judgment. We feel that very few clinicians, however knowledgeable they may be in their own specialty, develop proper radiological judgment. The limited field of their own practice places limits upon radiological experience. We feel that the clinician who tries to interpret his own films places himself in the position of the radiologist of a couple of decades ago, who of necessity learned painfully by trial and error. We do also readily acknowledge and explain to the student the limits of radiology.

We spend a good deal of time in teaching students to fill in a request for radiological service properly. To some extent, this is a thankless task. Our experience shows that most students do this very well for the first two or three months of their internship. After that many interns, even bright men, tend to become careless. However all in all, the time spent in teaching this is worthwhile. We tell the student that when he gets into practice he will almost surely acquire patients. As a doctor he should demand for his patient the best of care, with as little expense to him as the circumstances permit. One way in which he can do this is to plan carefully what he

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requires in x-ray service. This is to be written down plainly so that no error can occur.

We stress the necessity for stating on the requisition exactly what is wanted. Errors in requests are pointed out. A broad example would be the difference between asking for "Spine" when what is required is "Lumbo-sacral spine". Another example would be "Barium Swallow" when what is wanted is an examination of the "Stomach and Duodenum". Still another is a request for the examination of the ankle, when the injury is obviously in the foot. We go into this very thoroughly and in much detail. We also stress the great need for giving a working diagnosis, history and signs and symptoms, if prompt reporting is to be expected. In many cases the history is absolutely essential in arriving at a diagnosis. We feel that we are making headway in this, and that other hospitals and radiologists and their patients will benefit by such instruction of our students.

We tell our students that they should not tell the radiologist what films he is to have made. The doctor should say what part he wants examined and what is to be looked for. Telling a radiologist what films are to be made is equivalent to asking an internist to consult with regard to a patient, and explaining to him that he is to percuss and auscultate the chest.

The students are warned about bothering the technicians, interfering with established routine and persuading them to leave out certain views that may not at the time seem important. The students are told that most radiologists leave their technicians entirely alone when they are working. The radiologist, from experience, has laid down a routine for the technicians to follow. Only in certain cases will the radiologist interfere with this routine. The technician works more accurately when left alone. Experience has shown that if routine procedures are not carried out, the cost to the patient, not only in money but also in physical disability, may be high indeed.

We teach our students to be careful about ordering needless x-rays. We tell them that experience shows that the new practitioner orders many more x-rays during his first year or two in practice, than he does subsequently when he is treating many more patients.

We demonstrate to students the reasons why portable x-ray films should be requested only when absolutely necessary. The limitations of the portable machine put radiology back twenty years. Many portable films are useless and others may easily lead to wrong conclusions. Films made in x-ray rooms are so much superior that only real necessity should cause portable films to be made. Portable films also cost more.

It is during the student's final year when he is a "shadow" in the department (an important period in his training), that we teach him many of these things, and about practice, and ethics, and medico-legal problems, and all those affairs that revolve around the practice of radiology. We teach our students to be careful and considerate in their handling of old people; to not carry out for little purpose expensive and uncomfortable procedures which only upset the patient and make his final days less happy. We like a nice balance between scientific curiosity and humanity.

I like to tell the students about coming out of a large Canadian hospital one day. I had just seen many patients being put into extremely uncomfortable positions and having all sorts of things done to them for better or for worse. I was in a philosophic mood as I walked down the street. I wondered whether all that doctors do to people was always necessary. Then, as I passed a church I saw displayed this text—

> "In God have I put my trust: I will not be afraid
> What man can do unto me."

It is my fond hope that old graduates of Queen's who take the time to read this paper will remember some of these things I once talked about to them. That they will, having acquired more experience in the ways of medicine and medicine men, be in a better position now to appreciate these teachings is perhaps not too much for which to hope.

CLINICAL NUTRITION BRIEFS

Review of the feeding of extreme premature infants at the Chicago Lying-In Hospital revealed that the 45% incidence of retrolental fibroplasia occurring in 1946-1949 was associated with formula feedings while previous and subsequent experiences with human milk or relatively dilute cow's milk formulas led to an incidence of only 7%.—*Nutrition Reviews*, 11: 103, 1953, Sodium Restriction and Retrolental Fibroplasia.

PROSTATIC SURGERY IN CANADA WITH AN ANALYSIS OF 222 CASES OF TRANSURETHRAL PROSTATECTOMY*

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WE MUST APOLOGIZE for presenting a subject which has been covered so frequently but it is one on which there continues to be great diversity of opinion – possibly because the prostate gland lends itself to a variety of approaches and, unlike any other organ, can be removed by attack along natural channels.

It can be truthfully claimed that the advances in prostatic surgery have not lagged behind those in any other field. Open operations are now exact procedures. Hæmostasis and infection are controlled better, convalescence is rapid, and mortality is low.

Though the urethral approach has been long employed it is only about twenty years since really effective instruments have been available for this method of attack and still more recently that any large number of surgeons have become proficient in their use. It is now an exact procedure. It is possible in most cases to do as complete a prostatectomy as it is by any other method and to control effectively hæmorrhage and infection.

In order to present a picture of the present situation in Canada, we collected statistics in 1951 from a representative group of teaching and non-teaching hospitals. The total number of transurethral operations recorded was 5,930; suprapubic operations 1,527; retropubic operations 593, and perineals 10. The latter number is so small that we can disregard this procedure altogether. We have not added our own figures as we do almost all cases by the transurethral route and they will be dealt with separately.

In teaching hospitals, prior to 1951, 62.1% prostatectomies were done by the transurethral method with a 3% mortality, 37.6% by suprapubic methods with a mortality of 5.5% and only 0.2% by the retropubic method. In 1951, these figures were: transurethrals 74.8% with a 1.2% mortality; suprapubics 11.4% with a 5.7% mortality and retropubics 9.8% with a 5.5% mortality. The figures in non-teaching hospitals are not greatly different except that there were fewer retropubic operations. I am sure, however, that in hospitals in smaller cities the great majority are done by the suprapubic route but it is not possible to get accurate figures from these places. We have collected a further group of figures for 1952. An analysis of these shows the situation to be virtually unchanged. It is clear then, that an increasing majority of cases is being done by the transurethral method.

It is widely believed that the type of operation depends on the indication in each case, that the operation should be made to fit the patient and that, therefore, the surgeon should be proficient in every method of doing prostatectomies. This is a very large order and we doubt that many hospital staffs would be sufficiently proficient in such a variety of postoperative care. It would be more reasonable to say that the surgeon should be proficient in any two of the methods.

Though avowed resectionists, we admit there is good reason for the argument that it is better to do an open operation in large glands and in younger men. For a time we considered such a course and, when a prospective case came along we would decide to make the final decision after passing a 'scope—but in every instance the gland came out before the 'scope. Now, when we want to do an open operation, we never dare look in first. We have done six suprapubic prostatectomies in the past four years.

In order fully to evaluate the transurethral operation we have made a critical analysis of the hospital records of 222 consecutive cases which were done in the latter part of 1950, and the first four months of 1951. We also sent a questionnaire to these patients and an analysis of 165 is presented. Space does not permit a complete tabulation or discussion of all the findings, so we will deal only with the most important considerations.

We were interested in checking our clinical judgment against the microscopic findings in the tissue resected. There is a discrepancy of 4.5% but there is no doubt that a considerable number of those we said were malignant will prove to be so, despite lack of histological proof. This is, of course, a common experience and is not due to the fault of either the clinician or pathologist but to the site of origin of the disease close to the capsule. The percentage of clinically malignant cases was 17%, of which 15.5% were proved histologically.

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The size of the prostate on rectal examination did bear a close relationship to the urethroscopic findings. This is, of course, true only if the bladder is empty when the rectal examination is made. There were 29% reported as large on digital examination; there was marked elongation of the posterior urethra in 25%, and gross trabeculation of the bladder in 28% of the cases. There was a similar relationship in moderate and small prostates.

We have not been careful in gathering up all tissue, so that our figures as to the amount removed are not reliable but in the majority of cases the weight of this tissue is between 25 and 50 grams.

An analysis of cases re-operated upon shows that obstruction and infection were the main reasons. In some cases the obstruction was so slight it was difficult to be certain whether or not it played an important part. While it is a fact that you can usually go back and remove more tissue from any patient who has been resected and, indeed, from some who have had other operations, we were surprised to find that the majority of these cases that we re-operated upon had had small glands and now had some slight contraction of the vesical neck, a "nubbin" of prostate, or a stricture of the urethra. We were not able to remove much tissue from any of them. This refutes the contention that the small glands are best for resection. While, technically, this may be true they certainly do not give the best results. In fact, we are considering doing them by some competitive method! When the patients with really large glands return with further trouble they come back with bleeding, rarely with obstructive symptoms.

The strictures we now see are not of serious degree, and we rarely see any of the anterior urethra. We are satisfied that this is owing to greater care to avoid trauma and the use of smaller sheaths if the urethra is tight. The F. 28 sheath had been used in 75% of the cases. We considered it necessary to use the smaller sheaths in 25% of this series. We now use the F. 26 sheath in 75% of our cases. We do not perform external urethrotomy.

For years we felt that vasectomy to prevent epididymitis was essential but have now abandoned it entirely. In this series in which it was not done we had only six cases (2.6%) during their hospital stay. It was not always easy to be sure from the replies but we decided that 29 cases (18%) had suffered from it after returning home. One would certainly have had an erroneous impression from hospital incidence alone. Considering the fact that there are two possibilities of epididymitis in every case it might be argued that the actual percentage was only half the figure given!

We also endeavoured to determine how many patients had fever after leaving hospital. There were 27 (17%) cases who replied in the affirmative to the question about chills and fever. In this instance, too, considerable ambiguity made interpretation of the answers to this question rather difficult. As one often sees in history taking, a patient's impression of chills and fever tends to err on the side of exaggeration. Nevertheless, it was apparent that more than a few cases in this series had transitory bouts of pyelonephritis, though in the majority of cases the fever was associated with epididymitis.

Despite the fact that we use tap water as an irrigating medium we have never observed hæmoglobinæmia after operation and we have looked for it. Recently we had our first case of oliguria. It was fatal. We believe that overdistension of the bladder and laying bare wide areas of the prostatic capsule are probably important factors contributing to the entrance of irrigating fluid into the circulation. We use only low spinal anæsthesia, and try not to have it too profound. We feel that this is important so that the patient will be aware of any of these occurrences or any other undue trauma and the surgeon must be gentle in all his manipulations.

We used blood transfusions in 9.5% of our cases. This is far less than many of our friends in urology and in other branches of surgery commonly employ, but we have no reason to believe any of the complications which we have had could have been avoided or our morbidity improved by use of more blood. In this, as in other details of treatment, we depend more on clinical judgment than laboratory tests.

An analysis of the bleeding, turbid urine and irritative symptoms follows closely what one would expect from a study of urethral convalescence, in that the great majority of patients are well within a month.[1] This is the time that epithelization of the vesical neck is complete. It is only now that one would expect urine to become clear, and while chemotherapy in the earlier period is useful in controlling infection, it is at this time that most dramatic results in clearing

up pyuria are seen. We believe forcing fluids is actually more important during the first few weeks than chemotherapy in patients with kidney damage.

There was a significant number of patients who continued to have some bleeding, frequency or turbid urine and yet had never bothered consulting anyone about it. If this group had had chemotherapy or had a sound passed many would have got well much sooner. Winsbury-White has made the same observation regarding passage of sounds following suprapubic prostatectomy.

The majority reported the stream as satisfactory. Eleven per cent described it as "super" and 3% poor. In most of these latter cases there is probably some degree of stricture or remaining prostate. We have occasionally seen a case with a poor stream and yet we can find no reason to account for it.

We used no medication in 8% of cases. In half of the rest we used sulfonamides and the other half antibiotics. Any day that temperature rose to 100° or more was considered a day of morbidity. Judged on this basis 26.5% had no morbidity and 54% not more than three days.

As is usual with all methods of prostatectomy persistent infection was the cause of the majority of unsatisfactory results. This may be due to an infected upper tract or to an unsuspected diverticulum; complications which obviously would have no relation to the type of operation but, most frequently, it is due to persistent obstruction either at the vesical neck or in the urethra. In Shiver's analysis of over 14,000 cases this occurrence was highest after one-stage suprapubic prostatectomy (4.6%) and multiple resections (4.5%). It occurred in 2.7% of cases who had a single resection. He states that prolonged pressure of packing or bag hæmostasis following enucleation is one of the commonest causes of sepsis in the immediate postoperative period and quotes Nesbitt: "Trauma to the urethra is the commonest cause of infection in the immediate period following resection". This, of course, predisposes to stricture later. We think these observations are most important and must never be overlooked.

While the question on sexual ability was not put in for frivolous reasons, it did produce the most interesting replies and could have formed the basis of a separate paper. In 8% of cases there was an improvement and, in 61%, it was as good as before; 12% of cases who had some function before now had none at all. In the main these figures are not too dismal since they do include the carcinomas. One of these, age 69, was potent six months after resection, orchidectomy and stilbœsterol! One man of 45 was able to impregnate his wife.

Incontinence, at one time the most feared complication, is today the least of our worries. There was not a single case in the 1951 series in spite of the fact that it included approximately 15% carcinomas. We did, however, have two cases in the last two years who had really troublesome incontinence—one was a carcinoma.

The average hospital stay was 10.8 days, through 34% were out within a week. There are always a few cases which spoil one's figures and we have had our share. There were five deaths, a mortality of 2%. In our last series of 607 cases we had twelve deaths which is also about 2%. We do not believe that we will ever be able to improve on this figure.

CONCLUSIONS

1. Despite the very real advance in open operations on the prostate gland, the great majority are being done by the transurethral method in Canada.

2. In the analysis of our transurethral surgery the most troublesome complications were the result of infection and persistent obstruction. We do not feel that we would have had any less trouble if we had done a larger number of open operations.

3. Such a study brings to the reviewer's attention the unsatisfactory results and we think is a real challenge to any surgeon to improve the quality of his work. We have decided that we must continue a complete analysis and follow up of all our cases.

4. There were 26% who said they had never had any frequency or irritation; 18% who never had grossly turbid urine, and 49% who never saw any blood after discharge. Thirty-four per cent went home within a week. We feel that this is clearly a group with which no open operation can compete and rather than dissipate our energy in trying to do half a dozen types of operations and keep our staff upset all the time, we should direct all efforts to enlarging this select group. We believe this is possible.

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EPIDEMIC JAUNDICE*
(VIRAL HEPATITIS)

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EPIDEMICS OF JAUNDICE have been occurring in Europe since 1761. They have apparently accompanied most wars since that date, including the American Civil War and World Wars I and II.[1] The civilian populations have also shared in these outbreaks, Germany and the Scandinavian countries having the greatest incidence. The Mediterranean littoral was the source of major epidemics in both world wars.

It is indeed remarkable how similar these epidemics have been. With few exceptions all the epidemics have been described as mild. The prodromal period is described as 5 to 10 days in duration during which malaise, headache, slight fever, anorexia and vague abdominal pain are complained of; this being followed by dark urine, and icterus of the scleræ and skin, recovery being complete in about 6 weeks.

A few minor variations are also described in the different reports, such as the appearance of jaundice without prodromata, or, the occurrence of prodromata without the appearance of jaundice. This latter observation is indeed very significant in the light of our present knowledge.

Major variations were generally misinterpreted. The severe fulminating cases resulting in death were considered unrelated to this disease, and were known as acute yellow atrophy of the liver.[2] Another variation, described by Weil in 1885,[3] where the disease was ushered in by chills and fever accompanied by jaundice and hæmorrhages into the skin and mucous membranes was considered a more severe form of the same disease. In 1914, however, during an epidemic of this type affecting 4,000 miners in Japan, Noguchi[4] was successful in isolating the causative spirochæte to which he gave the name leptospira ictero hæmorrhagica.

When the great outbreak of epidemic jaundice occurred in Gallipoli in World War I, neither this organism nor any other bacteria was found to explain the disease, and it soon became apparent that Weil's disease or leptospirosis was clinically and bacteriologically different from the predominant form of epidemic jaundice.

The pathogenesis of the mild form of epidemic jaundice was also confusing. Because the disease rarely resulted in death, there were very few autopsies. Virchow[5] in one autopsy described a catarrh of the duodenum and bile ducts with a mucus plug in the ampulla of Vater. He concluded that the jaundice resulted from biliary obstruction by the mucus plug. This resulted in the disease being named "catarrhal jaundice". So firm did this opinion hold that, despite subsequent observation by others to the contrary,[6] little advance was made.

With the re-entry of British and American troops into the Mediterranean littoral in World War II, epidemic jaundice rapidly re-appeared. Research teams of British and American armies promptly entered the field and by what now appears simple procedures the problem was solved. (1) By administering orally, fæcal material from patients suffering with epidemic jaundice, they were able to reproduce the disease, thus proving its infectious nature.[7] (2) By means of liver biopsy, a method previously used by Scandinavian workers but little known, they demonstrated beyond doubt that the jaundice was due to inflammation of the parenchymal liver cells, in other words, hepatitis.[8]

The result of these two observations, therefore, ended in the accurately descriptive name by which the disease is now known – infectious hepatitis.

The reproduction of the disease by oral administration of the fæces soon led to the discovery of the viral nature of the infecting agent, and many of the characteristics of this virus have since been identified.[9]

Coincident with the outbreak of epidemic jaundice in the East, another more serious form appeared in the West. In 1942, about three months following the inoculation of vast numbers of troops against yellow fever, thousands of cases of jaundice appeared among the troops of the armed forces of the United States.[10] Liver puncture studies showed inflammatory changes identified with those of infectious hepatitis.[8]

Such episodes on a smaller scale had previously occurred under similar circumstances, but were never understood.[11] It was soon established that pools of human blood, used in preparation of the yellow fever vaccine, contained the virus which when introduced parenterally into large

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numbers of troops resulted in a new, artificial form, of epidemic jaundice. This has become known as homologous serum jaundice or serum hepatitis.

Because of slight clinical differences in the orally and parenterally produced hepatitis, these viruses are not considered identical. The virus of the former, therefore, is being designated by the letters IH and that of the latter by the letters SH. Several strains of each are believed to exist.

It is, therefore, evident that epidemic jaundice is the result of a viral infection, either, (a) naturally spread by IH virus; or (b) artificially spread by SH virus.

The term epidemic jaundice is now used exclusively to denote viral hepatitis. Weil's disease is now known as leptospirosis.

The following is a brief review of the more significant or more recent developments in the study of viral hepatitis.

VIRAL HEPATITIS

Etiology.—(1) Human beings are the only known source of viral hepatitis.[12] (2) It is believed that there are two types of virus: (a) IH, which usually enters by the oral route, but may also be introduced parenterally, with a short incubation period, and accompanied by prodromata. (b) SH, always introduced parenterally, with a long incubation period (50 to 150 days), and almost no prodromal symptoms.

Epidemiology.—(1) Most evidence indicates that infectious hepatitis is spread through person-to-person contact, and most likely by the oral route. Because of seasonal incidence—autumn and early winter—the respiratory route has been considered as a method of spread, but this has not been confirmed experimentally. (2) Proven water-borne, milk and food epidemics have occurred.[13] (3) Transmission by biting insects such as lice, bed bugs and mosquitoes has not been proven. (4) The greatest incidence occurs in Germany, Scandinavia and the Mediterranean Basin. (5) In civilian populations, it more commonly affects children in the ages of 10 to 14 and drops remarkably after the age of 30. (6) The artificial spread of the disease by needle and syringe of both IH and SH hepatitis results from the asymptomatic carrier. SH virus has been found in blood of human volunteers during their incubation period. The virus may be present in such high concentration that 0.01 c.c. has induced the disease in volunteers.[14] One professional donor was the source of infection in four different recipients of his blood over a period of four years. It is not known how long the virus persists in the blood or fæces of recovered cases.[15] SH virus has not been recovered from fæces. (7) The virus cannot be eliminated, by the usual methods of sterilization, from blood for transfusion, without rendering it unfit for use. Certain weak strains of SH virus can be destroyed by ultra-violet light. The virus cannot be removed from water by the usual concentration of chlorination. (8) The virus is filterable, resists heat 56° C. for 30 minutes (SH 60 minutes).

Clinical course.—The incubation period of IH hepatitis is 16 to 30 days, that of SH hepatitis 50 to 150 days.[10] In the former, the disease usually manifests itself fairly abruptly with headache, malaise, fever (slight to moderate), anorexia, constipation and vague abdominal pain. This may last 5 to 10 days before dark urine, light stools and icterus appear. In many cases, and particularly SH hepatitis, these prodromata are absent and the patient may present with dark urine or icterus. The severity of the jaundice is very variable; pruritus at times is troublesome. The physical signs are, the icterus, a large tender liver, and often the spleen is palpable; occasionally posterior cervical nodes are enlarged. Usually by the end of the second week, appetite returns and jaundice gradually clears. By the end of six to eight weeks recovery is complete in about 85% of cases. Their clinical recovery has been paralleled by complete pathological recovery as determined by liver biopsy studies, and by return of liver function tests to normal.[16]

Fatal cases occur in 0.2 to 0.4% of cases;[5] as a rule anywhere between the fourth to the twentieth day. Without any previous premonitory signs, lethargy and coma, alternating with restlessness, excitement, and delirium are followed by death. The pathological findings in these cases are similar to those described in acute yellow atrophy with severe destruction of liver cells. Subacute forms in which death occurs in 3 to 6 weeks have been reported.

About 15% of cases show a more chronic tendency. Of these, relapse prolongs the convalescence but many recover completely within one year. A small number go on to a chronic form of hepatitis complaining of easy fatiguability, abdominal distress, intolerance to fat, and a large and tender liver. Whether true cirrhosis

develops in these cases is still undecided. Certainly liver biospsies have shown persistent chronic inflammatory changes. A few cases of persistent jaundice without any other impairment of liver function have been reported. The end result of all these cases will not be clearly determined until many more years have elapsed.

Prognosis. – Activity, when the total serum bilirubin is above 3 mgm. %, predisposes to prolonged convalescence.[17] The initial severity of the jaundice is no indication of a serious prognosis. Persistent symptoms over a long period of time, however, or frequent relapses after small amounts of activity may be serious. A persistently large or tender liver is serious. Persistent splenomegaly is probably serious.

If liver function tests deviate from normal over a long period of time the prognosis may be serious. In this regard perhaps the most valuable liver function tests are elevated serum bilirubin, delayed bromsulphthalien excretion, and abnormal flocculation tests. It should be emphasized that no one test and particularly the cephalin cholesterol flocculation test is in itself very significant. This latter test may remain abnormal for a very long time without great significance.

Abnormal findings with needle biopsies of the liver which persist for a long time, indicate serious damage. But here too considerable reversibility is known to occur.

PROPHYLAXIS

Infectious hepatitis (virus IH).—No widely applicable method has been found to prevent infectious hepatitis. The danger of infection exists essentially from the asymptomatic carrier, or the patient in the pre-icteric phase. It is estimated that for each case of infectious hepatitis with jaundice, there is at least one that never develops icterus. No test is at present known that will aid in the detection of the asymptomatic carrier. It would, therefore, appear that sound hygiene is still our most effective means of prevention.

Natural immunity, at least temporarily, develops after infection with each of the viruses (IH and SH) but infection with one does not result in immunity against the other.[18] Passive immunity with gamma globulin has been proven definitely effective in preventing infection in human volunteers.[19] Small epidemics have been aborted by its use.[20] No method of active immunization is as yet available. The use of gamma globulin in combination with an attenuated virus to produce active immunity is being considered.

Serum hepatitis (virus SH).—Serum hepatitis as a result of parenteral administration of infected blood or its products is now widely known. Its prevention, however, is not as simple as would appear at first glance. Potential infection is present in hundreds of biological products, immune sera and various vaccines, since many are made with human blood. Even simple procedures, such as vena puncture or skin puncture for blood counts, are sufficient to cause infection, if the instruments used are contaminated with the virus. With the long incubation period such minor procedures are forgotten by the time jaundice appears, and isolated cases of this type might well be mistaken for a sporadic case of infectious hepatitis.

Similarly, laboratory workers handling infected blood and stool are liable to infection through broken skin. In areas where hepatitis is endemic, most meticulous care in sterilization and handling, are essential in prevention of this disease.

As for transfusion services, every donor should be examined with special reference to liver disease. A donor, with a history of hepatitis within one year, should be rejected. The urine should be checked for bilirubin and increased amounts of urobilinogen, and after the blood is drawn, serum bilirubin and flocculation tests should be normal before the blood is released.[12] In choosing materials for transfusion, whole blood and single plasma units offer the least risk, whereas, large plasma pools impose the greatest risk.

The value of ultra-violet ray in eliminating the infecting agent from blood is as yet not definitely established.[12]

TREATMENT

Three major principles of treatment are of proven values: (1) Bed rest. (2) Diet. (3) Avoidance of further insults to the liver.

1. *Bed rest.*—Careful experiments carried out by Swift *et al.*[17] showed very conclusively that those patients who were active when their serum bilirubin was above 3.0 mgm. % had a more prolonged convalescence and a greater incidence of chronic liver disease than those who were kept at rest.

Certain criteria for allowing a patient to become active are recognized:[21] (1) At least 3 weeks of bed rest with bathroom privileges. (2) The liver should not be enlarged or tender. (3) The

patient should be free of symptoms, have a good appetite, and a sense of well being. (4) A normal serum bilirubin for one week, or, if this is slightly elevated a negative direct van den Bergh. (5) Return of symptoms or a tender liver, with activity, is an indication for further rest. Other liver function tests may be helpful in doubtful cases.

2. Diet.—High protein and high carbohydrate diets with moderate restrictions of fat is recommended. Butter is better tolerated than other fats. Methionine and choline have not been found of value.

3. The prevention of further insults to the liver are to be achieved by the avoidance of alcohol, surgery and anæsthesia, particularly ether, chloroform and ethylene. Where emergency surgery is imperative local or spinal anæsthesia is recommended.

The value of drugs is very limited. In the very severe, acute cases it has been felt that bacterial products from the intestinal tract contribute to liver damage. It is, therefore, suggested that aureomycin 1 gram every 6 or 8 hours orally, or, 0.5 gram every 6 hours intravenously should be used. The use of ACTH and cortisone have been found helpful by some observers in the severe cases, but this has not been generally confirmed.

SUMMARY

1. A brief historical review of epidemic jaundice showing the stages by which the etiology and pathogenesis were finally determined, is described.

2. A summary of our present knowledge of etiology, epidemiology, clinical course, prognosis, prophylaxis and treatment is presented.

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Case Reports

THYMECTOMY IN MYASTHENIA GRAVIS*

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THE SIGNIFICANCE of thymic enlargement or hyperplasia in myasthenia gravis is not clear. It has been claimed that in myasthenia the thymus is responsible for the interference with neuromuscular transmission either by the production of a curare-like substance[1] or by the inhibition of acetylcholine synthesis.[2] Marked improvement of myasthenic phenomena after thymectomy has been reported by various authors.

In Keynes[3] series of 137 patients, thymectomy resulted in moderate to complete remission in 87% of cases. Ross[4] has recently reviewed 100 consecutive thymectomized, non-tumour patients of Keynes, 7½ years after the operation. He found that 41% had no symptoms and required no prostigmine; 26% minimal symptoms and small doses of prostigmine; 20% some improvement still requiring prostigmine; no change in 6% and 7 deaths, one from myasthenia and 6 from unrelated causes.

In Viets and Schwabs[5] series the results of thymectomy indicate that female patients benefit more from the operation than males. In Eaton and Clagett's[6] series, thymectomy resulted in 50% improvement in cases with a thymus tumour, and 56% improvement in myasthenia

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patients without tumours. However, when they compared the results of thymectomy with controls treated conservatively, they found that the difference in favour of thymectomy was only 7%, which is not statistically significant, and from now on they operate on patients with thymoma only because of the potential malignancy of the tumour.

The variable results of thymectomy in the treatment of myasthenia gravis are difficult to evaluate.

An attempt to explore the problem further was made by Wilson and his associates.[7] The thymus glands removed at operation by Keynes from patients with myasthenia gravis were extracted and tested on the isolated nerve-muscle preparation. The neuromuscular blocking activity of 42 individual glands was assayed and as a control thymus glands of normal adults which were removed at autopsy were similarly tested. The results of Wilson's investigation indicate that the group of glands which had the greatest neuromuscular blocking activity on the isolated nerve-muscle preparation, produced on their removal the most beneficial clinical results. The second group showed little neuromuscular blocking activity, corresponding to the Keynes group of patients where the removal of the gland produced little or no effect on signs and symptoms. Control observation with the normal adult thymuses removed at autopsy showed only little or no neuromuscular blocking activity. Thus from Wilson's data, and from clinical observations it would appear that the choice of suitable myasthenic patients is of supreme importance if they are to benefit from the operation.

Careful studies by Viets suggests that the most favourable results after thymectomy are obtained in young females with a relatively short history of myasthenia and with a previous history of a remission. The operation should never be performed on a patient not properly controlled with prostigmine, and this drug must be given by continuous intravenous infusion during and for a few days after the operation. Ether must be avoided as it depresses the neuromuscular function and the endotracheal cyclopropane is the anæsthetic of choice.

This is the case of a 25-year old female whose myasthenia was first diagnosed 5 years ago. The presenting symptoms at that time were weakness of the extremities, and of the ocular and facial musculature.

She had been frequently hospitalized for the last few years because of generalized weakness with respiratory difficulties. On the whole her myasthenia ran a fluctuating course, and she had only one significant remission 2 years ago which lasted for 3 months. At no time did she take less than 8 tablets of prostigmine a day.

It was felt that this patient might benefit by thymectomy; she was not responding too well to prostigmine. At the time of examination she was 5 months' pregnant, but the remission of the disease frequently observed in pregnant myasthenia patients did not take place in her case; if anything she became much weaker and her prostigmine requirement fluctuated from 12 to 30 tablets a day. Pregnancy itself was not considered a contraindication to the operation. Successful thymectomies were performed by Keynes[8] during pregnancy.

After a rest at home the patient was admitted to hospital for 2 weeks before the operation during which time her general condition was carefully evaluated. This included the assessment of the disease itself, bronchoscopic and respiratory function tests. Following bed rest, during which her prostigmine intake became stabilized it was considered that the patient had improved sufficiently to withstand the operation, particularly as there was no respiratory weakness.

The patient's daily prostigmine intake was averaged, and this was given by a continuous intravenous drip during the operative procedure, and for 2 days postoperatively. An additional 15% prostigmine was administered during the operation. Thymectomy was performed through the anterior mediastinal incision, under endotracheal cyclopropane anæsthesia. Considerable care was taken to remove the thymus intact, and not to rupture the pleura. A lobulated thymic mass weighing 24 grams was removed at the operation. The remainder of the mediastinum was explored for some aberrant thymic tissue and was found to be normal.

Histological examination showed multilobular masses of small, round, deeply staining cells, which from a cytological point of view are identical with lymphocytes. These structures tended to be aggregated about the Hassall's corpuscles which were relatively large. Although from a microscopic point of view this gland did not appear unusual for an individual of this age, it is of interest that the acute thymic involution which occurs after the third month of pregnancy was not observed in this case.

An x-ray was taken in the operating room so that the presence of pulmonary complications could be detected immediately. This and the x-rays that followed were negative. At the end of the operation the patient began to breathe spontaneously. Postoperatively the patient was supervised very closely and this included half-hourly blood pressure recordings and changing the patient from side to side. The mechanical respirator was at the patient's bedside for immediate treatment of respiratory failure. In the days that followed the patient made an uneventful recovery from the operation, and was discharged home 3 weeks later. The operation had no deleterious effect on the patient's pregnancy which proceeded in a normal manner.

A few weeks after the operation the patient's prostigmine requirement diminished, and fluctuated from 1 to 6 tablets daily. It became quite obvious that her muscle strength was improved. She could talk clearly, the ocular manifestations of myasthenia disappeared and she was working daily in a store in addition to her household duties.

The patient proceeded to term and had a normal labour. She was delivered of a male infant weighing 6 lb. which lived for 1 hour only. The autopsy disclosed a tear in the tentorium cerebelli with cerebral hæmorrhage, but no other abnormality. Following her delivery the patient made an uneventful recovery and was sent home still taking not more than 6 tablets of prostigmine daily.

She is maintaining the improvement observed after thymectomy and can be classified as belonging to the group B of Keynes: virtually well; minimal symptoms; small doses of prostigmine.

ADDENDUM

The thymus gland from this myasthenic patient immediately after its removal was sectioned, and a small portion was preserved for histological examination. The remainder was grafted into the abdomen of a monkey.

Prior to the thymus graft the muscular strength of this animal was repeatedly tested by one of us (H.S.) using a muscle stimulator and a kymograph recording device. Apart from some questionable weakness on the third day after the operation, no muscle weakness was detected in daily muscle strength recordings for a period of 30 days.

We are grateful to Dr. J. D. Hermann, Senior Thoracic Surgeon, Ottawa Civic Hospital, for his invaluable assistance, and to Dr. C. A. Mitchell, Director, Animal Research Institute, Hull, P.Q., for the facilities connected with the experimental aspect of this case.

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NAPHTHALENE POISONING FROM THE INGESTION OF MOTH BALLS*

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VERY FEW CASES of naphthalene poisoning from the ingestion of moth balls have been reported in the American literature. In 1949, Zuelzer and Apt[1] reported four cases of this type of poisoning, under the heading, "Acute Hæmolytic Anæmia due to Naphthalene Poisoning". All of the cases reported were between the ages of 2 to 3 years of age and all recovered.

They carried out experiments with dogs and were able to demonstrate almost identical symptoms and changes in the blood of dogs fed with a quantity of naphthalene, as were observed in humans after the ingestion of moth balls. They briefly reported a few cases from foreign literature, as follows: Goetze in 1884,[2] Nash,[3] Heine,[4] Taylor and Russel,[5] Konar, Roy and De,[6] and Smillie.[7]

Goetze observed restlessness, delirium, twitchings and cyanosis in a patient who had received large amounts of naphthalene, 6 to 7 gm. daily for six days. On the seventh day the urine was black. Blood studies were not made. Nash briefly reported the development of urinary frequency, passage of black urine, diarrhœa, transient anuria and headache and abdominal pain in a boy, 13 years of age, who had eaten a single moth ball. Unfortunately no data on the blood were given. Heine described a woman who had taken seven consecutive doses of 0.4 gm. of naphthalene for the treatment of oxyuriasis. Dysuria, weakness, drowsiness and numbness of the extremities developed within two days. The patient passed black urine and her skin became icteric. Liver and spleen were palpable. The urine contained hæmoglobin and albumin. The patient recovered.

Taylor and Russel recorded anæmic, icterus and hæmoglobinuria in an African negro who had ingested two moth balls five days earlier. Konar, Roy and De described the case of a man aged 24, who had taken an unknown amount of naphthalene, mistaking it for candy. Two days later the patient became pale, icteric, and semicomatose. The hæmoglobin on the fourth day was 30%, the red blood count 2,410,000 and the white blood count 31,200. The urine was brown but gave a negative test for occult blood. The patient died, and naphthalene was demonstrated in the tissues, stomach contents and urine. The liver showed patchy necrosis.

Anæmia of hæmolytic type similar to that observed in our patients was reported by Smillie in Brazil, in four patients who had received betanapthol. Because a group of 77 other patients who had been given the same drug in comparable doses failed to exhibit anæmia, Smillie concluded that special pre-disposing factors were required for the development of toxic effects.

Moth balls are a common and easily accessible household article, believed to be more or less harmless. That they can produce a rapidly appearing toxic disturbance when absorbed by the gastro-intestinal tract is not generally known. A report of a case of naphthalene poisoning, fortunately with a non-fatal sequel, would appear to be justified.

A healthy male child of 2 years with a negative family history, while playing at home found a number of moth

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balls. He promptly chewed and swallowed several of them. No attention was drawn to the parents concerning this event at the time, by the female attendant of the child. The following day the child developed a fever of 101°, became listless, irritable and pale. He vomited several times and passed several pieces of unabsorbed moth ball in the stools. Later in the day the urine was observed to be dark, reddish brown in color, food was refused for supper and the child spent a restless night.

The following morning the temperature was 102°. The child appeared very pale and apathetic, vomiting was repeated and food refused. The urine presented a dark brown reddish hue. Admission to the hospital was advised. A diagnosis of naphthalene poisoning was made when the urine was found to contain few red blood cells and much hæmoglobin, with 3 plus albumen and a few granular casts. Jaundice was apparent in the skin and severe anæmia apparent. 600 c.c. of whole blood was given by slow transfusion. Shortly after the transfusion the child ceased to vomit, took fluids freely and spent a less restless night. On the third day the child was much improved, colour was good and the urine appeared less dark in colour. The child began taking an interest in toys and ate some food. The temperature was 100°. On the fourth day the temperature was normal, a few granular casts and one plus albumen with much less hæmoglobin was reported, on examination of the urine.

The child was discharged from the hospital on the fifth day after admission. The urine was normal in ten days' time. The child was apparently well and the jaundice had disappeared.

	1st day in hospital	*2nd day in hospital*	*4th day in hospital*
R.B.C.	2,140,000	3,400,000	3,620,000
H.G.B.	5.2 gm.	8.6 gm.	8.8. gm.
W.B.C.	23,450	18,000	8,850
Sed. rate	6	8	15
Differential	Polys. 47 Lymphs. 41 Fragmentation of cells observed	Polys. 69 Lymphs. 17	Polys. 23 Lymphs. 72
Blood urea	42 mgm.	32 mgm.	30 mgm.
Blood serum bilirubin	1.8 mgm.		1.6 mgm.

A case of naphthalene poisoning from the ingestion of moth balls is reported. Complete recovery took place following a period of marked systemic disturbance, which was characterized at the onset, by vomiting, prostration and pyrexia, later by rapidly developing hæmolytic anæmia, slight jaundice, leucocytosis and acute nephritis. Complete recovery ensued. Fatal cases have been reported in the literature.

Differential diagnoses from Lederer's anæmia, an acute hæmolytic anæmia of unknown origin, is difficult unless a history of ingestion of naphthalene is definitely known to have taken place.

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SMALL BOWEL OBSTRUCTION FOLLOWING SUBTOTAL GASTRECTOMY AND RETRO-COLIC GASTRO-JEJUNOSTOMY*

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IT IS THE INTENTION of this note to report a case of small bowel obstruction by internal hernia, one month following a subtotal gastrectomy and retro-colic gastro-jejunostomy. The intestinal obstruction was the result of all the small bowel, with the exception of the last 8″ of terminal ileum, passing through a hole produced by the anastomosis which was bounded by the afferent loop in front, the transverse mesocolon above and the posterior peritoneum behind.

F.M., aged 40, a labourer, began in 1939 to suffer dyspepsia of the duodenal ulcer type and later that year a perforation occurred. At this time a laparotomy was performed with simple patching of the perforation, with satisfactory results. The dyspepsia recurred in 1951 and continued intermittently until 1953 when the patient began to vomit copious amounts of undigested food. He had at no time noticed any melena nor any change of bowel habits. Full investigation confirmed that he had a duodenal ulcer with pyloric obstruction. Blood chemistry studies revealed he was in physiological acid-base balance. His Hgb. was 17 gm. and his R.B.C. and W.B.C. were within normal limits.

Laparotomy was carried out on June 19, 1953, through a right upper paramedian incision. Many extensive tough adhesions were separated and a duodenal ulcer with much surrounding inflammatory reaction was found in the first part of the duodenum, penetrating into the pancreas. The duodenum was separated from the pancreas and transected, the distal end was invaginated and a three-fifths subtotal gastrectomy of the Polya type was performed. The stomach stump was then drawn down through the avascular space in the transverse mesocolon, and anterior and posterior sutures used to suture the mesocolon to the stomach. Using a short (3″ to 4″) proximal loop, a retro-colic, isoperistaltic gastro-jejunostomy was performed.

The patient received 500 c.c. whole blood and 1,000 c.c. 5% glucose solution in normal saline while on the table. The following day 40 m./eq. of KCL was added to his daily intravenous requirement. The Levine tube was removed on the fourth postoperative day and by June 25, the patient was taking eight small daily feedings of any food. He was discharged from hospital on June 29, and although he did not resume his work as a labourer at once, he was doing odd jobs around a farm on which he lived and was gaining weight.

On July 29, following his late evening meal, he was seized with a sudden sharp, crampy pain in his right upper quadrant. The pain was very severe and the patient vomited. The pain increased in severity and more vomiting ensued, his family physician was summoned and he was readmitted to hospital. Examination revealed a "board-like" rigidity of the abdomen with maximum tenderness and splinting in the right upper quadrant. There were no palpable masses and auscultation failed to reveal any increase in bowel sounds. Pulse was of good volume, but 110 in rate. A serum amylase at this time was 32 units. W.B.C. was 9,700 and Hgb. estimation

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showed 13 gm. A Levine tube was passed and intestinal decompression started. X-ray of the abdomen, supine and upright, failed to demonstrate any free air in the peritoneal cavity, any gross distention of bowel, or fluid levels. Two hours after admission it was decided to operate. His temperature was 98, pulse 120 and respirations 20.

The right upper paramedian scar was excised and the peritoneal cavity re-opened. Transverse colon was freed of adhesions and the duodenal stump and anastomosis were examined. The small intestine which presented in the wound was dark in colour, and upon further exploration it was found that almost all of the small bowel had passed through the opening posterior to the short proximal loop of the jejunum, *i.e.*, through the space bounded by the afferent loop of jejunum in front, the transverse mesocolon above, and the posterior peritoneum covering the pancreas, behind (See Fig. 1).

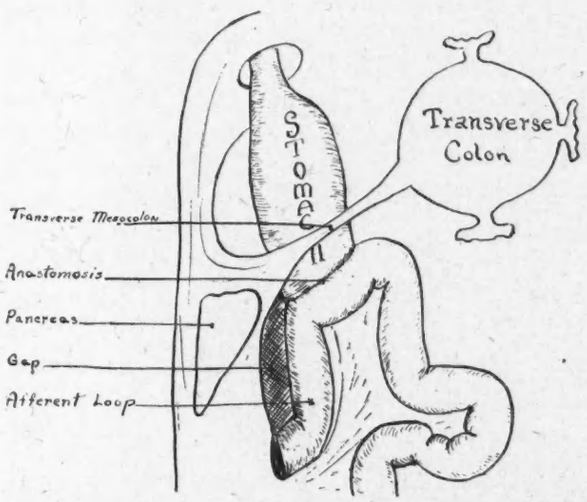

Fig. 1.—Lateral view showing gap and how it may close by stitching.

By gentle traction the herniated coils were easily reduced and almost immediately the discoloured portion became pink and normal. The gap was closed by suturing the visceral layer of peritoneum of the afferent loop to the posterior peritoneum over the pancreas.

The Levine tube was left in the stomach for three days postoperatively and fluid and electrolyte balance was maintained by venoclysis. On the sixth postoperative day the progress notes said, "a fully recovered man from every point of view", and from then on progress was uninterrupted. Follow-up notes to November 1953, report that he has resumed full work and is in excellent health.

In summary it may be concluded that this complication is obviously rare, putting it in the same category of obstruction as (a) herniation through the mesocolon following retro-colic gastroenterostomy; (b) slipping of a loop of gut round the left paracolic gutter following sigmoid colostomy; (c) similar occurrence round the right paracolic gutter following ileostomy. Experience has taught all surgeons that in order to prevent these obstructions these gaps must be closed at the time of primary operation. It is not difficult to obliterate this opening with a few stitches and thus by foresight avoid another remote, but possible postoperative complication.

ACUTE MYOCARDITIS OCCURRING IN BULBAR POLIOMYELITIS*

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THE FINDING of a myocarditis which could be the cause of cardiac failure in those patients dying in the acute stage of poliomyelitis is not a rare phenomenon. Recently, the literature was reviewed by Teloh[1] who was able to find 159 cases of fatal poliomyelitis, in which 72 had an acute myocarditis. In reviewing the material from his hospital he was able to add 47 cases of which 26 had an acute myocarditis. The incidence of myocarditis in fatal bulbar spinal poliomyelitis is 42% on the basis of this study. Jurow and Dolgopol[2] have recently stressed the frequency of this finding and also that of an interstitial pneumonia in such cases.

These reports have appeared for the most part in journals of pathology. It therefore seemed worthwhile to bring to the attention of clinicians the fact that myocarditis as a cause of death in poliomyelitis is more common than supposed.

CASE HISTORY

K.M., a 21 year old carpenter who had no history of important past illness other than recurrent chronic sinusitis was admitted to hospital on the fourth day of an acute illness which began abruptly with feverishness and a sore throat. The disease progressed rapidly with hoarseness, difficulty in swallowing, inability to cough effectively and difficulty in raising his abundant mucoid sputum. On several occasions he became alarmed by nasal regurgitation while attempting to swallow liquids. These symptoms were so severe that he had not eaten for three days.

On admission to hospital his temperature was 100° F., pulse 140 and respirations 18. Generalized body weakness was apparent on cursory examination. His whispering voice had a slight nasal quality.

Auscultation of the chest revealed numerous moist râles scattered over both lung bases. The breath sounds were bronchial in character in the same locations and air entry was variably impaired at both lung bases. There were no clinical signs of cardiac enlargement and the heart sounds were distinct and not remarkable. The heart rate was regular but rapid with a pulse wave of low volume rendering B.P. determination difficult: it was estimated repeatedly at 110/96. Although the patient was unable to raise his head there was no demonstrable neck muscle spasm or rigidity. Muscle power and tone were universally diminished in all his extremities and the deep reflexes were absent except for a minimal response from the right triceps. Both plantar responses were flexor in type and the abdominal reflexes were not elicited. Further examination of the patient at this time was not informa-

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tive. Early treatment with antibiotics and parenteral fluids was started. It was clinically evident that the patient was gravely ill and that his general condition was rapidly becoming worse. Shortly after admission the pulse rate had climbed from 140 to 180, respirations had quickened to 20 and he had become increasingly apprehensive. Notwithstanding these ominous signs, there was no indication of respiratory embarrassment, or of imminent circulatory collapse.

Laboratory investigations.—Hgb. 14.5 gr. %; WBC 21,000 per c.mm. with 91% neutrophils and 9% lymphocytes. Sedimentation rate was 5 mm. per hour. Urinalysis revealed a sp. gr. of 1.030, albumin 3 plus, Benedicts, green. The E.C.G. showed a rapid regular ventricular rate of 200 per minute with a normal sinus rhythm suggesting sinus tachycardia. The C.S.F. was clear and normal in pressure. Differential cell count on the fluid revealed 9 lymphocytes and 7 polymorphs per c.mm. Tests reported at a later date gave the level of protein at 25 mgm. %; chlorides 765 mgm. %, and the Lange curve was 0011100000.

Chest x-rays demonstrated extensive pneumonic shadowing throughout the lower two-thirds of the left lung field and to a lesser extent, throughout the right upper zone.

The clinical picturé and preliminary laboratory findings suggested poliomyelitis with bulbar and spinal involvement. Since he appeared to be deteriorating rapidly, the patient was transferred to the Isolation Hospital so that an iron lung would be available in the event of respiratory failure, but he died soon afterwards.

Autopsy findings.—Only the pertinent autopsy findings will be described. The heart weighed 375 gm. It was dilated, the dilatation being chiefly on the right side. No gross changes were seen in the myocardium.

Both lungs were voluminous. The right weighed 700 gm. and the left 750 gm. Throughout the remaining viscera there was evidence of congestion. In the brain, congestion of the blood vessels over the cerebral hemisphere was noted and after fixation the medulla and pons still appeared hyperæmic but grossly no areas of softening were found.

Microscopic examination.—In the medulla, at the level of the olive, there were histological findings compatible with a diagnosis of acute poliomyelitis. Necrosis of the nuclei represented by ghost-like outlines was present while others showed only an increase of their Nissl substance. There was a moderately heavy diffuse mononuclear cell infiltrate present which was found about degenerating nuclei, in perivascular situations, and in small collections in the subependymal regions of the 4th ventricle. The cellular infiltrate was such that it extended over the site of origin of the 10th cranial nerve and the respiratory centre. In the upper reaches of the spinal cord, these changes were found in the anterior horn cells and were even more marked. The cellular infiltrate was heavier and an extensive search was necessary to find a single horn cell which appeared to be viable. The inflammatory cell infiltrate extended to the meningeal surface. In the other sections of the brain, no evidence of an acute inflammatory process nor any neuronal degeneration was seen.

The myocardial findings were interesting. There was an acute diffuse interstitial myocarditis present in the auricles and the ventricles. This was represented by a moderately heavy infiltration of polymorphs and lymphocytes with a few macrophages intermingled in the interstitial fibrous connective tissue. This infiltrate did not appear to be concentrated in any one area, although its intensity did vary slightly from field to field and extended into the pericardium. No necrosis of myofibrils was noted in the sections (Figs. 1 and 2).

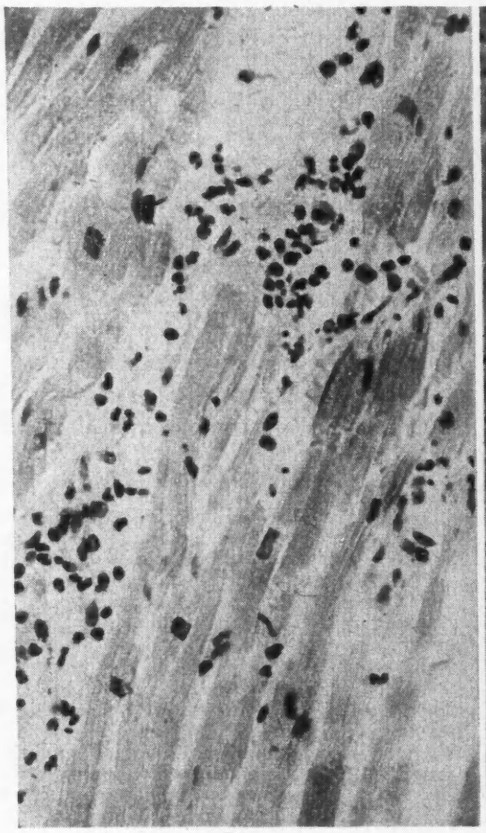

Fig. 1

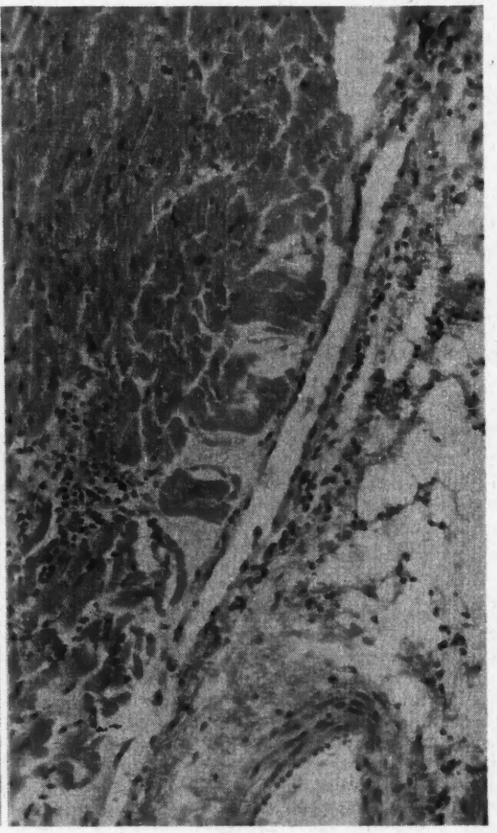

Fig. 2

Fig. 1.—Myocardium showing acute myocarditis without necrosis of myofibrils.
Fig. 2.—Myocardium with myocarditis and extension into pericardial fat.

In the sections of lung there was a considerable amount of œdema fluid in the alveoli. The bronchi contained small numbers of polymorphs and some of these extended into the surrounding alveoli so that an early bronchopneumonia was present. Moderate numbers of red cells were also seen in the alveolar spaces. No interstitial pneumonia as reported by Jurow and Dolgopol[2] was seen.

DISCUSSION

Clinically, paralysis of the soft palate, pharynx and vocal cord resulted in difficulty in swallowing and accumulation of the secretion in the oropharynx. These findings indicated involvement of the 10th cranial nerve and sections of the medulla showed that this cranial nerve and the area of the respiratory centre were affected. At the present time there is no agreement as to whether these changes are due to the virus of poliomyelitis or whether they might result from secondary hypoxia due to the respiratory difficulty. Clinically, the chief symptoms when the respiratory centre is involved, are irregularities in rhythm and depth of respiration. There are long intervals between inspirations and the respirations tend to be shallow. This patient's signs and symptoms did not fit exactly into the common pattern of respiratory centre involvement and it raises the question of the importance of the myocarditis found at autopsy. Usually poliomyelitis patients with an acute myocarditis have a florid appearance, the pulse is extremely rapid and difficult to palpate, and the blood pressure varies from low to slightly elevated levels. In such patients, there is a severe hæmorrhagic pulmonary œdema although this can vary in individual cases. Focal necrosis is usually seen in the medulla in the medial ventral reticular substance although the myocarditis is a more prominent finding. In this case while there are signs of bulbar poliomyelitis there are features of myocardial failure such as pulmonary œdema and the dilatation of the right heart. The findings of an acute myocarditis would be the basis for explanation of latter observations. This raises the possibility that there may be a specific viscerotropic type of virus responsible for the changes noted.

SUMMARY

The authors have reported a case of bulbar poliomyelitis in which there was an extremely rapid pulse with circulatory failure and in which at autopsy there was an acute diffuse interstitial myocarditis as well as the changes of bulbar poliomyelitis. Whether this lesion has been overlooked in the past or is the result of a different strain of poliomyelitis virus cannot be determined at this time.

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GOITRE IN A PREMATURE INFANT*

L. N. PEARLMAN, M.D., *Ottawa*

THE MATERNAL INGESTION of antithyroid drugs has been followed by the occurrence of goitre in both the newborn animal[3] and human.[2, 4, 5] In the latter, prematurity has been noted at least five times.[1, 5, 8] Associated congenital anatomic defects of the nervous system[4, 9] and of the heart[8] have also been mentioned.

Sulfonamides have also been implicated in the production of goitre in the post-natal period.[6]

In the following case, examples of several of these facets of the goitre problem occurred.

The mother was 26 years old para i grav. ii. Because of hyperthyroidism she had been taking 150 to 200 mgm. of propylthiouracil daily for the previous year. This dosage was occasionally doubled for periods of 3 or 4 days depending on "how nervous I felt". For about one month prior to delivery she increased it to 300 mgm. daily. She had also been taking Lugol's solution 8 to 12 drops daily for the year prior to delivery.

She developed hydramnios and spontaneously went into labour July 23, 1953 producing a female of 34 weeks' gestation weighing 4 lb. 1½ oz. The baby was born with a diffusely enlarged thyroid gland which was firm with small nodules. The head was held in retraction.

Her general condition for the first day of life was only fair. This gradually improved over the next two days. Thereafter, there was no sluggishness, excessive drowsiness or unusual variation in body temperature.

At age 3 days she began to have occasional trouble swallowing. The thyroid had enlarged a great deal and become firm and entirely smooth. The infant was put on 2 drops of Lugol's daily.

The next day she suffered a severe temporary blue spell. For the following 48 hours her breathing was irregular and further difficulty was encountered with feeding. She required frequent suction and a high oxygen atmosphere.

It was now realized that the enlarged gland was probably a thyrotropic effect.[3] Desiccated thyroid substance gr. ⅛ b.i.d. was therefore started at age 5 days. During the next week improvement both generally and locally was satisfactory. The gland became much smaller and the head retraction disappeared. At age 15 days the iodine was discontinued.

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During the next 10 days the gland varied slightly in size. By age 36 days all swelling had disappeared. The thyroid extract was stopped. However, a diffuse systolic murmur at the base of the heart was discovered. Since the baby was in no apparent difficulty, she was discharged home on August 28, weighing 5 lb. 3 oz.

The limited laboratory investigations which could be carried out were as follows: *Whole blood* cholesterol at age 5 and 13 days read 175 and 178 mgm. % respectively. These values are elevated for this age.[11] *Plasma* cholesterol at age 26 and 33 days were 145 and 155 mgm. % respectively—normal to slightly elevated values.

X-ray of the skeleton at age 32 days showed bone age to be within normal limits.

On September 8 the baby was seen again. She had a slight exophthalmos which the mother had noticed for the preceding week. The child seemed restless. The thyroid isthmus was palpable. The cardiac murmur was more localized towards the third right interspace. The rate was 178. There was no evidence of heart failure. Her weight was 5 lb. 14 oz. and serum cholesterol 166 mgm.

On September 12 the baby developed a series of blue spells and was again admitted to hospital. The only positive physical findings were the tachycardia and the murmur. Occasionally the baby showed a questionable very slight cyanosis.

X-ray and fluoroscopy of the heart, chest and skeleton revealed no abnormalities. An E.C.G. showed a probable sinus tachycardia with a rate of 172 to 180, C.T. 0.10 and a left axis deviation. It was ultimately felt that the heart might be presenting a subaortic stenosis with a further possibility of septal defect.

During the next 2½ weeks the rate varied from 140 to 204. The higher readings *seemed* to coincide with the occurrence of a mild diarrhœa and low grade fever and on one occasion with a cellulitis of a finger.

Because of the persistent diarrhœa she was given sulfadiazine and sulfasuxidine one grain of each q.4 h. daily from September 16. On September 24 because of the spread of the cellulitis erythromycin was added, 90 mgm. daily.

On September 27 the baby developed severe head retraction and again a diffusely enlarged thyroid. The pulse rate was 140. The sulfonamides were immediately stopped. Within 24 hours the retraction vanished and much of the swelling subsided. The baby was discharged home two days later with a palpable but not excessively large gland.

DISCUSSION

Although most mothers treated with thiouracil give birth to normal infants, about 20 to 30% in selected series, apparently show some effect of the drug on the fetal and neonatal human thyroid.

It has been observed that in mothers receiving iodine with the thiouracil, no abnormalities of the offspring occurred.[4] This case negates this observation. In fact goitre has occurred in infants whose mothers have received iodine only.[10] On the other hand, it is felt that thiouracil derivatives should be discontinued in the last month of pregnancy to avoid trouble.[9] This case would not have benefited from this approach since the baby was born a month prematurely.

It has been stressed that the avoidance of a myxœdematous condition in the mother is important in the prevention of fetal goitre.[1, 2, 5, 7] The obstetrician should bear in mind that during pregnancy the B.M.R. is *normally* elevated, even as early as the third month.[7] It is possible for thiouracil derivatives to produce a *relative* hypothyroidism. Administration of the drug in amounts required to produce a B.M.R. normal for a non-pregnant woman, would in effect be producing a degree of hypothyroidism during pregnancy. This pitfall may be circumvented by giving the drug for short periods only[5] or by the more rigorously controlled regimen outlined by McGavack.[7]

With regard to the infant's goitre, the original improvement might have occurred without medication, since this is not an unknown phenomenon. The exophthalmos at age 7 weeks has also been described.[2] It is not a permanent effect.

The recurrence of the goitre at age two months could conceivably be due to the sulfonamides but its rapid regression makes this a speculative concept, which is strengthened perhaps by the alterations in size observed during the first weeks of life.

The tachycardia observed in the second month of life may have been an iatrogenic thyrotoxicosis, but its persistence off and on for one month after the thyroid extract was stopped makes it a doubtful etiologic agent, particularly since the blood cholesterol remained somewhat elevated. The infections the child suffered may have been factors, but this cannot be the whole story, since the pulse rate varied despite the persistence of the infections.

It is most likely that the last series of blue spells were due to the tachycardia which in turn was probably associated with the congenital heart.

SUMMARY

1. A case of congenital goitre complicated by congenital heart disease in a premature infant is presented.

2. Attention is directed to the prophylactic control of maternal thiouracil administration and the possible influence of sulfonamide ingestion.

I am greatly indebted to Dr. W. C. McIntosh for the opportunity of seeing this case and for data relevant to the mother's history.

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Clinical and Laboratory Notes

A METHOD OF DIRECT GASTROSCOPY*

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IN SPITE of improved facilities, the establishment of a definite diagnosis of a gastric lesion in certain cases is still a real problem. X-ray, while of invaluable aid, often brings us to the point where a questionable shadow throws grave doubts as to the presence or absence of a serious lesion that makes prompt diagnosis mandatory. The gastroscope, also of great assistance, has, in many cases, failed to bring about the final diagnosis. Biopsy at time of laparotomy has in many instances proven the only real way of establishing such diagnosis. However, in the case of early lesions of the stomach, the biopsy must often be made from within the organ and this entails either a punch through a gastroscope or gastrostomy. The comments to be made simply outline a procedure that I found of considerable help in these obscure cases.

Under general anæsthesia a small left paramedian incision is made. Whether this incision be right or left paramedian depends a great deal on the operator's choice. The stomach is then inspected and palpated looking for the suspected lesion. If the lesion is obvious then the necessary surgery is carried out. If there is some question about the presence of a lesion or the nature of a lesion the field is covered with a drape such as used for spinal anæsthesia, in other words, a large drape with a relatively small round opening. Through this two intestinal Allys forceps are placed about an inch and a half apart on the anterior wall of the fundus of the stomach. A small incision is then made in the longitudinal axis of the stomach. The tip of the sterile sigmoidoscope is then introduced through this small opening. The opening should be just large enough to afford a snug introduction of the instrument. With the sigmoidoscope in place, a small quantity of air is injected by means of the bulb syringe, and with the stomach partially dilated the entire organ from the œsophageal opening down to the pyloric opening is open to inspection. I was surprised at first to find that the injection of a small quantity of air will balloon out the stomach making examination of the viscus a simple matter. Any suspected areas can also be directed to the opening of the sigmoidoscope by the hand.

Examination of the cardia and œsophageal opening is particularly interesting by this method of examination and should be of some value in case of suspected œsophageal varices. Examination also affords an adequate means of obtaining a biopsy either with a punch in the less accessible regions or direct dissection of tissue in the areas more easily handled. The examination being completed, if no evidence of disease is found, the scope is withdrawn and the small opening is readily closed in the usual manner.

Of the various sigmoidoscopes available I find that the instrument in which the source of light is near the eye piece is preferable to those in which the light is at the distal end of the scope.

I should like to briefly review two cases in which this examination proved helpful.

The first case—of over three years' standing—was that of a woman in which a suspicious shadow was reported at the cardiac end of the stomach suggestive of a lesion. Gastroscopic examination was then performed. The gastroscopist reported that he was unable to visualize this particular area of the cardia by gastroscopy. Examination carried out by the procedure outlined proved that the suspected area was due to a particularly heavy rugal fold. Time and further examination have confirmed the absence of any neoplastic or other gastric lesion in this case.

The second case was that of a patient in whom a definite gastric ulcer was found and the question as to malignancy arose. In this case a biopsy was readily obtained and being reported upon as non-malignant a sub-total rather than a total gastrectomy was carried out.

I believe the chief advantage of this examination is that a larger area of the stomach can be visualized through the scope than by the ordinary gastrostomy and secondly because of the relatively small opening in the stomach the field of examination is almost bloodless. The inflation of the stomach also increases the scope of the examination.

It is realized that this examination has very limited application but the subject is reported in the hope that it may be of some help in these occasional obscure cases in which the presence or nature of a lesion is not clarified by the usual methods of examination.

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Editorials

THE CENTENARY OF QUEEN'S MEDICAL SCHOOL

There has been a peculiar pleasure in helping to prepare the present issue of our *Journal*. It is designed to celebrate the centenary of the founding, and the hundred years' accomplishment, of a great Canadian medical school. The occasion has rightly been recognized as one of much more than local or provincial importance. Queen's has contributed to the making of Canada, and we are glad to add to the national recognition of its high place in Canadian medicine.

The Journal itself has always enjoyed an extremely happy association with Queen's Medical Faculty. If we are to foster the incalculably valuable atmosphere of an editorial family it will be by developing such relationships as have grown up between our editorial management and our Queen's contributors. It was entirely natural for Dr. Eldon Boyd to suggest last year that such a commemorative number might be planned; it was just as natural to arrange for it. The resulting contributions have fully justified our expectations.

It is unusually pleasant to welcome to our editorial column Dr. Austin Smith, both as a Queen's graduate and as the editor of our great American sister Journal. This is the first contribution to our editorial columns by the editor of *The Journal of the American Medical Association*. It could not be more welcome, nor more peculiarly fitting.

Our Journal adds to the many expressions of goodwill which Queen's Medical School is receiving and will receive, its own congratulations and warmest wishes for continued success.

A WAY OF LIFE

Life at a university is a way of life unto itself. Life at a medical school is no exception. For several years the student sits apart, in effect, from the rest of the world and yet always he is being taught how to live with others. From morning to night he is subjected to studies which leave him little time for observing first-hand community life away from the campus. The few hours he can devote to pleasure are limited; he has few places of appeal, he has too little time to cultivate social acquaintances, and often he has insufficient funds for other than a temporary glimpse of pleasurable pursuits. To some degree, he leads an isolated life for three-fourths or more of a year and for the remaining time may be working to accumulate enough money to return to his isolated existence.

How, then, can a man or woman who goes through so many years of such existence emerge suddenly as someone trained to assume a rôle in community life and to exercise control over life and death?

The development of the medical student's background begins in his home, or his community, early in life. What determines a person's choice of a professional career is not always clear, especially to himself. What influences him most profoundly on the college or university campus often is vague and indescribable. The rôle of his family, his friends, his teachers, his classmates may be so indefinable that he asks himself in puzzlement sometimes "Why am I interested in this? Why am I like this? Why am I here?" Unusual indeed is the person who can say truthfully "This is what I planned in every detail."

There must be, therefore, a way of life, sometimes indiscernible but always influential. It is less evident to the younger graduate than to the older. It often is overlooked even by the teacher or the university official. And yet, there it is—more of an invisible form of pressure than a guiding beacon.

Many speak of the university spirit. It is evident in all universities worthy of the name. Queen's is no exception. In fact, this spirit seems to be unusually prevalent, so it has been said repeatedly, among Queen's men and women. The reasons may not be apparent but probably include the comfortable size of the school, the type of community in which it is located, the

calibre of its teachers, the liaison between teacher and student. This is no large city within itself. It is a campus and everyone on it is aware of this fact. There may be some who say that university acclamation is directly proportional to size. Such a view is of course without support, and rightly so, in the opinion of the true educator. Of equal importance is the realization of the graduate as he grows older that size and value of a product do not necessarily have any relation to each other. The more important things are not measurable in material terms, and include sense of responsibility, sense of appreciation, humility, respect, determination, outlook on life, attitude towards one's fellow man. These are difficult things to teach; they are more likely to be acquired. Some never are for various reasons.

A university provides tools, so to speak, for the graduate. They can be used to achieve success in the ordinary sense of the word or personal satisfaction, or both. But behind their use must be training based not on technological data nor sociological studies but on factors responsible for the attitude of the user. This is influenced, or even molded, by those responsible for college or university training in its broadest sense. When followed, and only when followed by experience, the mold which was shaped becomes hardened and the graduate student becomes not a filing cabinet of facts but a human individual.

So it is with Queen's University Faculty of Medicine. It would be easy to be biased on behalf of Queen's; after all, I am one of its graduates. But in all fairness, I must admit that while Queen's as a university has meant much to me, what it stands for has meant even more. And I can see this attitude in its teaching staff, a loyal industrious group widely known for its educational and research contributions. I can sense it also in the graduates as I meet them all over this country and not infrequently elsewhere in the world. I can sense it, too, in the voices of non-Queen's graduates who say "Oh, you're a Queen's man". One has reason to be proud in such moments.

But lest it be believed that I think only the Queen's man is due such recognition, let me say I mention Queen's because I know it best. What applies to Queen's applies to other educational centres and every man and woman should be thankful for the opportunities afforded him through college experience and for which his fees paid only a part. When I attended medical school I understood that my tuition fees paid for about one-third of the cost of my education. According to the latest report of the Principal of Queen's University the student is still a long way from paying for most of his education.

Most of us accept the life of a university without questioning the length of this life. It is only when anniversaries appear that there is sudden and often surprised recognition of this long span of service. When the special occasion is a centenary there is more apt to be true appreciation of what a university has contributed to a community, to a country, to a way of life. It is then that graduates realize what has been made possible for them by the long line of graduates preceding them. It is then they vow more than ever to try to repay in some way for the knowledge shared so freely with them. It is then they say "I am proud of my school" and mean it. Today, there are many of us spread throughout the world who are saying "We're proud of Queen's. We're proud of what it taught us. We're proud of what it stands for. It's our way of life".

AUSTIN SMITH

Chicago, Ill.

QUEEN'S MEDICAL CENTENARY NUMBER

This issue of the *Canadian Medical Association Journal* is presented as a Queen's Medical Centenary Number to the medical profession of Canada. Canadian physicians are hereby reminded that in 1954, the Faculty of Medicine in Queen's University at Kingston, Ont., completes one hundred years of service to Canada and Canadians. Some fifteen hundred physicians, now serving Canadians in villages, towns and cities from Newfoundland to British Columbia, were trained and disciplined in the limestone buildings on the Queen's campus and in the hospitals of Kingston and, more recently, Ottawa. Queen's medical graduates are aiding the development of the Canadian nation in medical education and research and in the medical services of Canadian armed forces, government, commerce and industry.

As part of the plans to commemorate the one hundredth birthday of the Faculty of Medicine

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at Queen's University, Dr. G. H. Ettinger, Dean of Medicine at Kingston, proposed that one number of the *Canadian Medical Association Journal* might be devoted to articles written by officers of instruction in the Queen's Faculty of Medicine. Arrangements for the publication of a Queen's Medical Centenary Number were made by Dr. Eldon M. Boyd of Queen's University and Dr. H. E. MacDermot, Editor of the *Journal*. The limitation of space has prevented publication in this number of all articles submitted by members of the Queen's Faculty of Medicine. Dr. Austin E. Smith, Editor of the *Journal of the American Medical Association*, who obtained his medical education at Queen's University and his initial postgraduate training in the Department of Pharmacology at Queen's University, was invited to write the guest editorial.

At the end of one century of service, the Faculty of Medicine at Queen's University is engaged in plans to improve further its facilities for Canadian medical research and for training young men and women in the science and discipline of Canadian medicine. The quality of medical service given to the Canadian people depends primarily upon the quality of medical education and research in Canadian medical schools. This responsibility is recognized and accepted by the Faculty of Medicine at Queen's University.

E.M.B.

Editorial Comments

GYNÆCOLOGY IN GENERAL SURGICAL TRAINING*

Much is heard these days about the return to general practice. An even greater need on our expanding frontier and in the smaller settled communities with hospitals, is for the trained general surgeon.

The general surgeon of fifteen years ago could shell out a prostate, do a total hysterectomy, treat an open fracture, do ordinary abdominal surgery, and even decompress a skull,—referring cases that were beyond him to the specialists at the university centres. Such men are becoming rare. Little attempt is now being made to train men on such a broad surgical basis.

One after the other the various surgical specialties have gone off on their own, till only what is left is regarded as the field of the so-called "general surgeon". One main branch, gynæcological surgery, has been split off and has been placed with obstetrics to form the comparatively new specialty of obstetrics and gynæcology. This in the writer's opinion (and he is no relative of Tom Cullen of Baltimore) is not in the best interests of surgical training in Canada.

So-called surgeons are being turned out in North America with no training in female pelvic surgery. Obstetrics is a basic subject and in the writer's opinion should stand majestically alone. It does so at Johns Hopkins Hospital. Abortions belong in such a department.

The combining of obstetrics and gynæcology into a specialty with the insistence and blessing of the Royal College, is turning out a city specialist who very rarely continues to practise extensively in both fields. A recent visit to a great American university found the Professor of Obstetrics and Gynæcology acting as a consultant only in Obstetrics. He had a heavy operating list. At the same hospital, the general surgeons were not permitted to do operative gynæcology.

The Mayo Clinic stands out as a bulwark against this general trend, and maintains the tradition of the true general surgeon. Its gynæcological surgery is done by competent general surgeons.

It has been our practice at the Ottawa Civic Hospital to get the senior surgical interns over to the gynæcological service and coach them through a total hysterectomy.

In the writer's opinion, gynæcology should be returned as a section of the department of surgery. Failing this arrangement, a compromise could be reached, by dividing the gynæcology services in our large hospitals into A and B divisions, A to go with obstetrics and train obstetricians and gynæcologists, and B to go with surgery and participate in the training of the genuine general surgeon. Regardless of where the gynæcology ward is fitted into the hospital scheme, the surgical interns of senior rank should swing over on to it for a portion of their training.

A year of general rotating internship followed by a two year rotating surgical internship, including gynæcology, would turn out a young surgeon better trained than most of his elders. Such a man should be granted major surgical privileges in all except perhaps the teaching hospitals. He would be a tower of strength as a surgeon in the smaller Canadian hospitals, and especially the more remote ones, where a full panel of surgical specialists is out of the question.

N. M. GUIOU

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*Contribution to the Queen's Medical Centenary Number of the *Canadian Medical Association Journal* from the Department of Obstetrics and Gynæcology, Faculty of Medicine, Queen's University.

THE DEAN'S MESSAGE AND GREETINGS FROM SISTER UNIVERSITIES

QUEEN'S MEDICAL CENTENARY YEAR

G. H. ETTINGER, M.D.,
Dean, Faculty of Medicine,
Queen's University

Queen's Medical School is completing its hundredth year. A hundred years is a small part of the life of a university; but it is so much more than human expectation that those who celebrate the centenary of a medical school are likely to examine the institution for signs of age. Buildings erected when the school was young may be subject to progressive modification, but there comes a time when no structural change can satisfy contemporary needs. Teachers serve their generation well, but inevitably retire to be replaced by men with different training and with appetites for newer (and expensive) equipment, both for teaching and research. It becomes the responsibility of the medical school to provide not only good teaching, but the facilities for its exposition.

Queen's is sensitive to this responsibility. In the past fifteen years it has added to its teaching and research facilities, but not sufficient for its current needs. Its teaching staff has been renewed with young and vigorous men. Many of these are ardent investigators who enjoy such confidence of research-granting bodies that they bring to the university, annually, nearly as much money for research as is provided by the university for its teaching program. These young men need additional space for their research, and for the modern methods of teaching in which they are well-schooled.

On the campus, the Departments of Anatomy and Physiology in particular are restricted in space, and extensions to buildings which house these departments are contemplated. This will provide an anatomical museum, small study rooms, workshops, small teaching and research laboratories, and departmental library space.

The Governors of the Kingston General Hospital have arranged to build the Walter T. Connell Wing, named in tribute to one who taught at Queen's for half a century. This will modernize the area in which medical students are taught, and will include classrooms, research and teaching laboratories, to whose cost the university feels obligated to contribute. Additional space must be added to the Richardson Laboratories Building, in order to accommodate the research program and graduate training program, and to yield proper facilities for post-mortem examinations. Additionally, there will be erected on the hospital grounds a large auditorium for medical school purposes, with common rooms and other facilities for student use.

For this needed expansion, the Provincial Government has already made some generous contributions to the hospital and to the university, but $750,000.00 is still required. It seems fitting that the university should appeal to its eighteen hundred living graduates during this, its centenary year, for liberal support for its building program. The canvass is already in hand, and many generous gifts have been acknowledged.

The week ending October 16, 1954 has been set aside as Queen's Medical Centenary Week. Invitations have been extended to all graduates to come at that time, to renew acquaintances, to participate in an excellent program of clinical and scientific merit, and to enjoy the festivities which will culminate in a formal university dinner, the annual medical formal, and an intercollegiate football game.

UNIVERSITY OF ALBERTA

In the century that has passed since the first medical students were enrolled at Queen's University, many changes have occurred in the methods and materials of medical education. The members of the Queen's Faculty of Medicine have taken a leading part and hold a high place in medical education and medical research in Canada.

On the occasion of the Centenary of the Medical Faculty, my colleagues and I, on the Faculty of Medicine of the University of Alberta, would like to express our warmest congratulations on past achievements and to express the sincere wish that over the future years the Medical Faculty of Queen's University will continue its valuable contributions of the past century and maintain the fine traditions which it has established.

JOHN W. SCOTT,
Dean, Faculty of Medicine

UNIVERSITY OF BRITISH COLUMBIA

In the past one hundred years, Queen's University Faculty of Medicine has witnessed and participated in a stirring panorama of medical progress. When it was opened in 1854, the "art" of bedside medicine was relatively advanced, but the fundamental sciences, upon which modern medicine depends so much, were just coming into their own.

In extending this greeting from the youngest medical school in Canada to one of the oldest and most illustrious, the University of British Columbia Faculty of Medicine pays tribute to

an institution which for a century has graduated splendid doctors. They have been trained thoroughly in the bio-medical sciences and well-versed in the arts and skills of bedside medicine. Their loyalty to their Alma Mater has become a byword. May "the Queen's spirit" continue to endure.

M. M. WEAVER,
Dean, Faculty of Medicine

DALHOUSIE UNIVERSITY

On behalf of the Faculty of Medicine of Dalhousie University I am happy to extend greetings to the Faculty of Medicine of Queen's University on the completion of one hundred years of teaching.

One hundred years of successful effort in the field of Medical Education is, in itself, no mean accomplishment but it is not merely for this effort but for its excellence that your Faculty has won fame. Your record is a proud one and those who know it realize that the celebration of this centenary will be but a prelude to greater accomplishment in the years ahead.

H. G. GRANT,
Dean, Faculty of Medicine

UNIVERSITE LAVAL

Le Doyen et les Professeurs de la Faculté de Médecine de l'Université Laval ont appris avec joie que la Faculté de Médecine de l'Université Queen's célébrera en 1954 le centième anniversaire de sa fondation. A l'occasion de cet heureux événement ils sont fiers de se joindre aux représentants des facultés sœurs de toutes les Universités canadiennes pour présenter aux autorités de la Faculté jubilaire leurs respectueux hommages et leurs sincères félicitations.

Au cours du premier siècle de son histoire, la Faculté de Médecine de Queen's par la valeur de son enseignement et l'importance de ses travaux de recherche a rendu à la profession et à la science médicales des services exceptionnels qui lui ont mérité l'estime et la gratitude de toute la population canadienne. Avec le témoignage de sa vive admiration et de sa cordiale amitié la Faculté de Médecine de Laval est très heureuse de lui adresser ses vœux les meilleurs de succès et de prospérité.

CHARLES VEZINA,
Doyen de la Faculté de Médecine

UNIVERSITY OF MANITOBA

I am instructed by the Faculty of Medicine at The University of Manitoba to extend heartiest greetings to the Faculty of Medicine at Queen's University on its Centenary. This event marks the completion of one hundred years of continuous progress and achievement in the academic field. During this time Queen's University has not only added many illustrious names to Canadian Medicine in teaching, research and the medical specialties, but has also produced a large number of general practitioners who have maintained the highest standards of practice throughout their professional careers. At its Centenary, the Faculty of Medicine of Queen's University stands very high in the list of Canadian Medical Schools, and will continue to do so by virtue of its progressive attitude toward medical education and the exceptionally high calibre of its teaching staff.

L. G. BELL,
Dean, Faculty of Medicine

McGILL UNIVERSITY

The Faculty of Medicine of McGill University, the oldest of the Canadian Schools of Medicine, welcomes the opportunity of sending a message of greeting to our colleagues in the Faculty of Medicine of Queen's University on the occasion of their Centenary Celebration.

Since our two schools serve adjoining constituencies of the Canadian people, we have had many opportunities to appreciate the sound training Queen's graduates in Medicine have received. Both of our Universities are Canadian expressions of the abiding interest of the Scottish people in higher education and this has served to create an additional bond between us.

We are happy to join our sister institutions in rendering just tribute to Queen's University for her past achievements and in expressing our confident expectation of even greater accomplishments in the years that lie ahead.

G. LYMAN DUFF,
Dean, Faculty of Medicine

UNIVERSITE DE MONTREAL

Dans son effort incessant pour se renouveler et se dépasser, la médecine crée d'abord l'apprentissage, puis l'école, puis, prenant conscience de ses affinités avec toutes les disciplines de l'esprit, s'associe avec elles dans le cadre d'une Université, et s'organise en Faculté.

Au Canada, pays jeune à croissance rapide et parfois tumultueuse, ces étapes de l'éducation médicale se succèdent en quelques décades, dans la première moitié du 19ième siècle, le long de cette voie d'eau qui servit de véhicule à sa naissante civilisation. A Montréal d'abord, puis, s'échelonnant à distance égale, à Québec, à Kingston, à Toronto, des facultés de médecine se constituèrent.

Et c'est ainsi qu'au "Queen's College" de Kingston, une faculté de médecine naquit, il y a un siècle cette année. Plus heureuse que beaucoup d'autres, elle ne connut pas les étapes parfois pénibles de la métamorphose d'une école autonome en faculté universitaire. Elle subit ensuite des vicissitudes et des crises de croissance. Son histoire même démontre que, dans l'ère moderne, l'enseignement médical ne s'épa-

nouit pleinement que dans son milieu naturel, l'Université.

A la centenaire Faculté de médecine de "Queen's University", la Faculté de médecine de l'Université de Montréal offre des félicitations ainsi que ses meilleurs vœux de succés et de prospérité.

WILBROD BONIN,
Doyen de la Faculté de Médecine

UNIVERSITY OF OTTAWA

The Dean, the Faculty and the Students of the Faculty of Medicine of the University of Ottawa unite in offering their congratulations to the Faculty of Medicine of Queen's University on the attainment of the hundredth year since its foundation.

They recall the eminent distinction of its services to the advancement of medical sciences, and the brilliant achievements of its graduates.

Canada owes a singular debt of gratitude to your School for the renowned services it has rendered to our country in the field of medicine and in that of social progress. Your history, struggles, spirit and aims are an inspiration to all who labour in the same field, and the surest guarantee of your continued progress.

A. L. RICHARD,
Dean, Faculty of Medicine

UNIVERSITY OF SASKATCHEWAN

Greetings from one of the youngest to one of the oldest! It is fitting that all who assume responsibility to educate the physician of tomorrow acknowledge their debt to the pioneers of yesterday. At Queen's, early faculty members, university administrators and teachers at "The Royal of Kingston", solved knotty problems relating to the faculty itself, university trustees and the practising profession. The enthusiasm of the founders survived stormy early years and the school grew up in robust clinical tradition. A substantial Scottish flavour was added thereby to the practice of medicine throughout the land.

In later decades, with the flowering of scientific scholarship at the university, Queen's won prestige in academic medicine. Laurels in teaching and research led to attainment of that hallmark of maturity—a reputation for growing and exporting professors. Perhaps in the next era she will join forces with other universities to mobilize medical education in the study of the health needs of Canada.

We wish you satisfaction and joy in your second century of accomplishment.

Cha gheill! Cha gheill! Cha gheill!

J. WENDELL MACLEOD,
Dean, Faculty of Medicine

UNIVERSITY OF TORONTO

It is perhaps no exaggeration to say that in the early part of this century most of the students attending the country schools in the Ottawa valley and the counties of southeastern Ontario, when they thought of university education, believed that this meant going to Queen's at Kingston. Often indeed the school-masters in those solid stone schoolhouses in Lanark County with the neatly painted signs S.S. No.—, who guided the destinies of the farmers' children, had spent a year or two in the storied halls of Queen's and were staying out to teach so that they might save sufficient money to return and finish their degree. The students who sat under them were unaware whether they had been to any College of Education to learn the art of teaching, but at least one of them remembers such teachers with affection and appreciation.

My own first knowledge of Queen's Medical School was through such teachers and among them the occasional one who was reading for his medical degree. Due to the restless, migratory tendency of the early years of the century, perhaps reflecting the spirit of the Scottish settlers who had come to Lanark county some two generations earlier, I did not even see the town of Kingston until many years later. In the meantime, however, I have come to know something of the influence of Queen's University and its Faculty of Medicine in the development of Canada. Queen's can indeed look back with satisfaction and a pride in the achievements of the past century. Successive generations of teachers have preserved their heritage of the Scottish tradition of medicine on which the school was so firmly established.

The Faculty of Medicine in the University of Toronto salutes its sister Faculty of Queen's on the occasion of the Centenary celebrations of 1954, and rejoicing in her pride of tradition and achievement, offers its congratulations, high hopes and confidence in her future.

J. A. MACFARLANE,
Dean, Faculty of Medicine

UNIVERSITY OF WESTERN ONTARIO

It is a great pleasure to join with sister Faculties of Medicine of Canadian Universities in bringing a message of congratulation and high tribute to the Faculty of Medicine of Queen's University which now completes one hundred years of service. Queen's University and its Faculty of Medicine have played a most important rôle in the educational and professional life of Canada over a long span of years, and no doubt will continue to extend and expand this service in the future.

Congratulations on past performance and best wishes for the future from the Faculty of Medicine of The University of Western Ontario.

J. B. COLLIP,
Dean, Faculty of Medicine

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Men and Books

THE ASTONISHING CAREER OF JOHN PALMER LITCHFIELD

By the late THOMAS GIBSON, M.A., M.B., C.M.(Edin.), *Kingston, Ont.*

WHEN SET TO THE TASK of tracing the early history of the medical faculty of the University of Queen's College by the late Dr. A. D. Blackader of Montreal, the writer's curiosity was aroused by the name of John Palmer Litchfield, who volunteered to teach forensic and state medicine as one of the five lecturers appointed by the Board of Trustees in 1854. The following year he was chosen to take charge of the criminal insane in their new quarters upon the estate of Rockwood recently purchased by the government of the United Canadas from the heirs of the late John Solomon Cartwright. The records of that institution were found to contain the somewhat startling statement that Litchfield had been appointed Inspector of Hospitals for the colony of South Australia in the year 1839.

Soon after this discovery, a note appeared in the *British Medical Journal* upon a paper by Dr. Alfred Lendon of North Adelaide, So. Australia, concerning the development of hospitals in that colony. Therein the fact of Litchfield's inspectorship was confirmed. This led the writer to correspond with Dr. Lendon, who kindly lent him a copy of a paper upon the career of Litchfield in So. Australia. No light, however was thrown therein upon his antecedents. After another interval, Dr. W. W. Francis suggested that search be made in Morgan's *Bibliotheca Canadensis* of 1867. Here was found Litchfield's account of himself in considerable detail.

The writer offers himself as guide over the trail of his own research. This seems to be the best way to sustain the dramatic suspense which constitutes the piquant allure of all such enquiries.

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The Colony of South Australia was proclaimed for settlement in December 1836, and its capital was named Adelaide in honour of King William the Fourth's Queen. Early in the year 1839, the barque *D'Auvergne*, of 440 tons burden, out of London, Captain P. Le Huguet, master, arrived at Adelaide with immigrants and cargo. On board were John Palmer Litchfield, ship's surgeon, and C. W. Litchfield, his brother, who rose to the position of an Inspector of Police.

Some time before setting out, John Litchfield had met Mr. Gouger, the secretary to Governor Gawlor of South Australia. He appears to have offered himself as physician and naturalist for the colony. Gouger who replied that the reported appointment of Litchfield to the staff of the Westminster General Dispensary opened a far more flattering prospect than any the Antipodes could offer. In spite of this, however, and with no definite expectations, Litchfield set sail.

Having arrived at Adelaide, he announced himself in the *Adelaide Register* of May 25, 1839, in the following terms:

> "J. P. Litchfield, M.D., etc., late Physician to the Westminster General Dispensary, Lecturer on Forensic and State Medicine at the Blenheim School of Medicine, and Physician to the London Infirmary for Diseases of the Skin, has the honour to inform the public that he has commenced the practice of his profession as a consulting physician in Adelaide. Dr. Litchfield takes this method of announcing his intention on account of the enquiries which have been made upon the subject."

In his correspondence with the Governor, he claimed, in addition, to have been one of the lecturers recognized by the Royal College of Surgeons, and by the Society of Apothecaries, London.

In the official *Gazette* of June, 1839, it was announced that Litchfield had been appointed Inspector of Hospitals for the colony without salary. The only hospital at the moment was a small thatched cottage, scarce weather-proof, and lacking the most elementary appliances for the care of the sick. It was managed by four of the colonial officials, of whom the colonial chaplain was chairman. When he resigned this post, Litchfield took it in virtue of his Inspectorship. In September of the same year, Litchfield, claiming to be a Fellow of the Linnaean Society, delivered a lecture upon the flora and fauna of So. Australia, with much éclat.

In the spring of 1840, he was discovered horse-whipping his erstwhile friend, Mr. Gouger, in the ante-room of his office. A certain Captain Tolmer, rushing to the rescue, handled Litchfield rather roughly. The latter apologized to Mr. Gouger, but exacted from Tolmer a similar solatium.

Litchfield was for ever writing to the Governor suggesting plans for the protection of the health of the colony. Such were the provision of a constant supply of potent vaccine, the revenue which might accrue from breeding medicinal leeches in a tank in the Torrens River, the need of a public abattoir, and, above all, for a hospital worthy of the capital. He was active in promoting schemes for raising money when the Governor decided to build.

When the hospital opened its doors, Litchfield proposed that he should be given a salary now that there was something to inspect. He also offered to attend the medical cases in the public wards. He was much chagrined when he discovered that the Inspectorship was to be abolished, and that the governing board of the hospital was to include no member of the medical profession. He thereupon proposed to start a private asylum in which he offered to care for

the pauper insane of the colony for five hundred pounds a year. The Governor seemed at first to favour the plan, but would not promise more than three hundred pounds. Litchfield rashly rented two houses in North Adelaide, the second as a convalescent home, thus involving himself in serious overhead charges. He had but two private patients to start with.

About this time the Governor came to the conclusion that this stranger had been protesting too much. He made search, therefore, and found no evidence, in Great Britain or Ireland, that Litchfield had taken a qualifying course of medical studies, and that his name could not be found upon the roster of Fellows of the Linnaean Society. A Dr. John Charles Litchfield had, in November, 1825, been admitted a Fellow, but had been rejected within the year for non-payment of entrance and maintenance fees.

In June, 1841, Mr. Gouger wrote to Litchfield that an act was about to be passed legalizing private lunatic asylums, provided they were under the control of persons possessing such diplomas as would be recognized in Britain. At present, however, the colony could not afford to pay for the care of pauper insane in private asylums. In his reply Litchfield gave himself away rather badly by pointing out that in Britain it was not necessary to be a qualified practitioner to run such an asylum, and that he had already arranged that two physicians in Adelaide should certify all his admissions. If he had himself been qualified, one other opinion would have sufficed. On the back of the last of Litchfield's letters to the Governor in regard to this matter the secretary wrote:—"When the proposition was mentioned the Governor smiled and said that he could not place government patients under Dr. Litchfield's hands. 'He is not a regularly educated medical practitioner, but got his degree at Heidelberg (by purchase)'." This last suggestion must surely have been offered by Litchfield himself as a forlorn hope. The pauper insane were left in the basement of the penitentiary under the care of Dr. Nash, the new colonial surgeon.

Towards the end of July, Litchfield found himself in prison for debt. The act of Victoria recently passed in England in relief of such cases had been endorsed by the colony, and his petition for release came before a judge on August 20. He is described therein as John Palmer Litchfield, proprietor of the lunatic asylum of Moorcroft House, North Adelaide. The judge satisfied himself that Litchfield had practised in Adelaide and that offers had been made him which had led him to believe that he might succeed in his new venture. Soon thereafter Litchfield set sail for England and Australia knew him no more.

Recent enquiries by correspondence with the Rector of Heidelberg University make it clear that purchase of a degree was impossible in those years. Regulations governing conferring of degrees were revised in 1821. A *bona fide* honorary degree would have been on record.

It was not until Henry Morgan's *Bibliotheca Canadensis* was consulted that light was cast upon Litchfield's earlier and later career. He provided Morgan with these details in the year before his own departure "from out this bourne of time and place."

The following is a free rendering of Morgan's memorabilia: Litchfield was for a long time connected with the news and periodical press of Great Britain, and is still correspondent for a leading London journal. In 1834, he wrote the introduction to the *Library of Popular Instruction*, London, and in 1835, *Outlines of Geology* for that library. In 1935 he published lectures upon skin diseases in the *Lancet* and *Medical Gazette*, London, was physician to the Infirmary for Diseases of the Skin, and one of the staff of the Westminster General Dispensary. In 1836-37, he was a contributor to the *Monthly Chronicle*, the *New Monthly Magazine and Humorist*, and *Bentley's Miscellany*. In 1837-38 he was a director of the metropolitan newspayer company, and one of the editors of the *Constitutional*, London. In 1839 he emigrated to South Australia, where he edited for a time the *Adelaide Independent*, and was Inspector of Hospitals for the Colony. From 1842 to 1844 he was in Paris as correspondent for the *Courier* and *Court Chronicle*, both of London. In 1844-45, he was in France, Belgium and Holland as correspondent for the *League*, London. In 1845-52, he was superintendent of the Walton Lunatic Asylum, near Liverpool. In 1845, he published *Essai sur la Raison et la Folie, thèse inaugurale*, Dewsbury, and contributed papers on the therapy of insanity to the *Museum* and *Medical Times*. (The *Museum* was not a medical journal. The files of the *Medical Times* have been examined at the Army Medical Library, Washington, without discovery of Litchfield's name in the index.) In 1853, he came to the United States, and was one of the editors of the *International Journal*, published in Boston, New York and Portland, Me. (Mr. Shapiro, of the Boston Public Library found Litchfield's address in the directory of 1853: "John Palmer Litchfield, physician, Invalid Food Office, 215 Washington Street". Editorials in the *International* were signed only with the initials of the editors; during that year some bore the initial "L").

In 1853-54, he was in Montreal, editing the *Weekly Pilot*. In 1855, he was appointed Superintendent of the asylum for the Criminal Insane at Rockwood, near Kingston, Ontario. He was for a time professor of Forensic and State Medicine at Queen's University. He was also agent for the American Associated Press. Here endeth Morgan.

Upon the whole then, in looking back over his career, it was his journalistic exploits which filled Litchfield's memories of the past. The inspectorship was a make believe, and his account of his eleven years as a medical professor is very incomplete.

With Dr. Francis' aid the lectures in the *Medical Gazette* were found. In the first, Litchfield reviews the attempts that had been made to classify these diseases, and goes on to demonstrate and discuss a case of scabies. A specimen of the infesting parasite was shown under a lens. It is a workmanlike performance, and the style of writing is good. In another lecture he drops a remark which explains a good deal. He praises the ability of Dr. Biett, whose practice at the Hôpital St. Louis at Paris he had observed at various intervals over two years. Thus he became

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adept in French and was able, in 1845, to write an essay on psychiatry in that language. Many years afterwards, in little Kingston, when Dr. John Stewart was promoting his claim to be appointed penitentiary surgeon, he made a strong point of his ability to speak French. "No other member of the staff," said he "could do this except Dr. Litchfield".

It would be easy for any young journalist, with a flair for medical studies, to pass into the wards or attend out-patient clinics. Dr. Biett's brilliant demonstrations would furnish forth many lectures upon skin diseases. Perhaps Litchfield's long connection with the press of London enabled him to have these five lectures printed in important medical journals. But why, when they read so well, were there only five?

Before the passing of the medical act of 1858, which established a Council of Medical Registration and Education in Great Britain, London practitioners used to offer courses of lectures to assist the somewhat irregular studies of medical students, and, incidentally, to bring credit to themselves. The records of the Royal College of Surgeons show that on January 29, 1828, one John Charles Litchfield, surgeon, Haymarket, applied for recognition as a lecturer but his claim was postponed until he could prove that he had given one successful course. The claim was not renewed.

The secretary of the Hospital for Diseases of the Skin, London, in reply to an enquiry as to whether Litchfield's name appeared upon their records, replied that it did not, and added the interesting information that even in 1938, men got away with strange impostures in London.

"We know one unregistered practitioner of dermatology, and it appears that he gets patients sent to him by practitioners. Not exery general practitioner is willing to spend some pounds on a medical directory, and most of them are too busy to look up a man in a library. The man I have in mind does practise, with the aid of a pharmacopœia taken from a skin hospital, plus a knowledge of skins picked up as a radiographer."

In *Bentley's Miscellany*, edited by Charles Dickens. Vol. III, 1858, pp. 504-506, there is a brightly written essay upon *The Postman*, enlarging upon his significance in the diversified human relationships of life. In the same volume, *Oliver Twist* was being serially continued from Chapter XX, with illustrations by George Cruickshanks. In the *New Monthly Magazine and Humorist*, edited by Theodore Hook, 1838, part first, pp. 511 *et seq.*, there is a long tale entitled *Wedding Tactics*. The scene is set in post-revolutionary Paris. In both these essays the author is named simply "Dr. Litchfield."

Some illuminating facts were brought to light by looking at the files of the *Weekly Pilot*. In the issue of June 7, 1854, the name J. P. Litchfield appears at the head of the leading articles, as editor. On June 21 appeared a review of the *Autobiography* of Benjamin Robert Haydon, the painter of historical subjects and friend of Keats. "We knew poor Haydon," writes the reviewer, "we attended his lecture upon the rôle of historical painting, and found that its tone of excessive abuse of the Academy and of several of the artists made it quite unfit for insertion in the press." A long article appeared on August 22 upon the need for urging upon the citizens of Montreal the claims of public health. "We recollect," says the writer, "twenty years ago hearing Mr. Farr lecture to empty benches upon Hygiene." Those were experiences of Litchfield's life as a London journalist.

His name appears on March 3, 1855, among those appointed by a local committee at Montreal to ask the Governor-General, Sir Edmund Head, to be present at the opening of the preparatory exhibition of commerce and the arts, from which objects would be chosen for the Canadian pavilion at the approaching Exhibition at Paris.

On March 12 he moved the adoption of the address to be presented to His Excellency at the close of the fair. He also moved the vote of thanks to the mayor. With the committee he was present at a luncheon given to the Governor at the Donegana Hotel. Finally, he signed at the head of the list of jurors, the awards to successful exhibitors. All this is quite in keeping with his pushfulness in Adelaide, but here he played a natural part as publicist and gave good service with speech and pen.

On May 15, 1855, there appeared a leader condemning the *Montreal Argus* for twitting the ministry because it had appointed Mr. Bellemare, editor of *La Minerve*, and Dr. Litchfield, editor of the *Pilot* to offices at the disposal of the government.

"With respect to Dr. Litchfield, it requires a genius like the editor of the *Argus* to make a job out of the affair. To constitute a job, an appointment must be given as a bribe. Dr. Litchfield has neither connections nor friends in power in this country; he has no political influence, nor has he taken a prominent part nor felt much interest in our local politics. He came to Canada bringing with him certificates from the most distinguished practitioners, as well of England as of the United States proving that he had much acquaintance with the treatment of the insane, to the study of which he had given much time and attention. His present appointment is the consequence."

This refers to his election as Superintendent of the criminal insane at Rockwood, near Kingston. His applying for this recalls his attempt to start a private asylum at North Adelaide in 1841, and his claim to have been medical superintendent of the Walton asylum near Liverpool, a small privately owned licensed house which was closed in 1861. He might well have been a lay manager there and picked up the little then known about the treatment of mental cases and of the practical management of such an institution.

On September 10, 1855, a letter was written to the *Pilot* over the signature "Fair Play", upon

the medical schools of Upper Canada, in answer to an article in the Toronto *Globe* which had contained the following remarks:—

"The Province must have a school of its own. At present, a high church establishment (Trinity) and a private institution (Dr. John Rolph's) are the only sources for the training of our youth in the medical profession."

"Fair Play" comments as follows:—

"Kingston, the second city of Canada West, has a medical school carefully organized in connection with the University of Queen's College, and this school is running a successful career, probably because it is devoted to the teaching of medical science irrespective of class or creed, and is free from the jealousies and rivalries which, unhappily, prevail in the medical schools in Toronto."

Litchfield is reporting upon his experience of the first tentative session of 1854-55.

It seems clear, therefore, that his major profession was that of journalism. Through it he obtained a footing in Boston and, with dramatic speed, a permanent post under Government in Upper Canada. He must often have marvelled at this good fortune when he cast back to those stormy years in Adelaide. His natural entry as an experienced pressman led him, in a fortunate year, into touch with the men of most influence in Montreal, then the seat of Government. His less flamboyant account of himself was accepted without question, and his readiness in amassing and dispensing information, the born journalist's gift, together with his ability to meet occasions as they arose, led him to three successive teaching offices and to a comfortable billet at Rockwood.

His first chair at Queen's was that of forensic and state medicine. The following session the lecturer upon obstetrics left town and Litchfield volunteered to teach this subject also. It was a full course, costing the student twelve dollars. Forensic medicine was a half course, costing six dollars. Fees were the only salaries in those days. After 1860 one man was permitted to teach but one subject, so the chair of obstetrics was given up. Later on, he taught institutes of medicine for a short period. He retired from academic work in 1865.

His lack of a regular medical degree came to be known, but he was not disturbed in his work at Rockwood. His regimen was characterized, according to the evidence of the son of his successor, Dr. John Robinson Dickson, by a pretty free use of alcohol by day and of sedative at night. Dr. Dickson was largely responsible for abolition of the routine use of alcohol in the asylums of Upper Canada.

Up till the year 1857 Litchfield lived on the corner of King and Lower Union Street. Then he occupied the North Lodge in the grounds at Rockwood. That handsome old Welshman, Mr. Thomas Evans, who laid out the grounds there, living himself at Portsmouth till well over his ninetieth year, remembered Litchfield as a kindly gentleman who used to drive around with his wife in a basket-bodied phæton.

Litchfield was interested in St. John's Anglican church at Portsmouth, and was a member of its first vestry. He was medical attendant to the family of Rev. F. W. Dobbs, by whom he was esteemed as a man of culture and a kind physician. He was for a time Captain of a company of volunteers.

After a lingering illness, due to heart disease, he died at Rockwood on December 18, 1868, aged sixty years. He was buried in the Hopkirk lot in Cataraqui cemetery. Twelve years later his wife died and his body was removed and laid by hers in the Anglican cemetery at Cataraqui.

On the day of his death there appeared in the *Kingston Times* a kindly obituary leader. Here, again, he is praised for kindness of heart, suavity of manners, and for his broad culture. Sympathy is expressed for the students of medicine scattered throughout the country in their sorrow over the death of "their favourite professor".

Will it be considered too fanciful if we suggest that John Palmer Litchfield was the son of John Charles Litchfield, surgeon, Haymarket, London? The latter, we may conclude, was a none-too-well-off practitioner, with a flair for botanical study, and a desire to achieve a fellowship of the Linnaean Society, the most august botanical fraternity in Europe. In this he failed for lack of a few pounds sterling. His sons were men of parts, but hampered in completing their education. John Palmer was entered as a dresser under the distinguished surgeon Mr. (afterwards Sir) Charles Bell of Middlesex Hospital. Prevented by circumstances of which we know nothing, he did not complete his apprenticeship nor obtain his degree. How soon he took up journalism we know not, but he must have early shown so much ability as led to his filling important posts on the Continent of Europe. And in Paris we know that he still followed after his first love—medicine—as opportunity presented itself.

It will be remembered that John Charles Litchfield desired to become one of the lecturers endorsed by the Royal College of Surgeons, London. In Adelaide, John Palmer Litchfield made groundless claim to have been a Fellow of the Linnaean Society and to have been a lecturer recognized by the Royal College. The possible relationship of father and son seems the simplest way of constructing this concatenation of facts.

The new-fledged group of lecturers to the twenty-three students who first read medicine at Queen's, contained a born surgeon of great ability—for a man who could excise an elbow joint with first intention healing four years before the birth of Listerian antisepsis was a rare

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phenomenon indeed. His name was John Robinson Dickson, an Ulsterman, student of Glasgow and graduate of the University of New York. He also took the degrees of M.R.C.P. and S. of London, and the F.R.C.S. of Edinburgh.

The anatomist had been a student of the famous and notorious Robert Knox of Edinburgh. His name was John Stewart, a Perthshire highlander who fancied himself of the royal line and sported the royal tartan on his professional rounds. He edited for two periods of years a paper called *The Argus*, at financial loss to himself and finally with a jail penalty for slandering therein one of his colleagues.

Perhaps most Odysseyan of the group, however, was John Palmer Litchfield, knight-errant of the pen and pestle.

GENERAL PRACTICE

THE COLLEGE OF GENERAL PRACTICE

CHAS. L. GASS, M.D., *Tatamagouche, N.S.*

THE GREAT and rapid broadening of man's field of knowledge has brought a consequent and necessary growth of specialism. Nowhere are these changes more evident, especially to physicians, than in the field of medicine.

One of the great forward steps in Canadian medicine in the past twenty years has been the establishment of the Royal College of Physicians and Surgeons for the training and certification of competent specialists in various restricted categories. But there has been some opposite reaction to this forward action. Time is needed to find and eliminate some weaknesses. We are not concerned here, although, perhaps we should be, with the adverse effect on the modern specialist who sacrifices breadth for depth: in some respects at least he suffers in comparison with the older type of specialist who worked his way to specialism through general practice. But the Canadian Medical Association has been concerned for some years with the effect which our modern advances with growth of specialism have had on the spirit and status of the general practitioner.

It is often stated that no man can keep up to date with all the new things in medicine. With the general advance the keen and hard working doctor has been struggling to keep pace and has been succeeding to a remarkable degree. He has developed in both breadth and depth in the past two or three decades, and perhaps that fact has not been sufficiently recognized. Increase in knowledge and efficiency has not been confined to the specialists' ranks in medicine. Yet there is a feeling in some quarters that the general practitioner is losing his status, not only in society but also in the ranks of the profession. One keen and forceful doctor from the West expressed this feeling rather succinctly when he said that he was tired of being told that he was the backbone of medicine and being treated as the coccyx! How well founded these ideas are is not clear, yet it is true that difficulties have arisen in some places which interfere with the practical pursuit of his calling by the general practitioner, especially in respect to hospital and teaching appointments. One reason for this is that there is no means of accrediting with high standing those general practitioners who by study and postgraduate work strive to keep up to date. They have no certificate of excellence in General Practice, such as the Royal College of Physicians and Surgeons grants to qualified specialists, to recommend their appointment. Whatever we may think of the real value of extra certificates, we must face the fact that today they seem to be highly desirable, and, in some situations necessary. Certainly, many general practitioners deserve some mark of distinction.

Is the general practitioner—the family doctor—losing his status in society? We live in an age which is not only materialistic but unbelieving. Perhaps the family doctor is not credited with the omniscience which his predecessor enjoyed. Perhaps his halo is getting a bit thin, but the growth of specialism is not to blame. "The fault, dear Brutus," lies elsewhere. What the people are asking for—almost pleading for—is more well trained, progressive, ethical family doctors, not more specialists. The demand for specialists, I believe, should, and does come from the general practitioners who need and appreciate their help. The general practitioner had better face the fact that the security of his status rests with himself.

There has been a great swing towards specialism since the war. The specialist rather than the family doctor has caught the fancy of many keen young medical students. Immediately post-war, the facilities for their training were strained to the limit. One hears the criticism that the medical schools and teaching hospitals are more interested in producing specialists than general practitioners, due to the influence and zeal of the Royal College of Physicians and Surgeons. In so far as this situation is real it is harmful to the profession. Certainly Canadian medicine would be much better if the general practitioner would exercise some of the influence and zeal which he undoubtedly possesses. There is evidence that the swing towards specialism has reached its peak and is in reverse. More young graduates are turning to general practice where they are needed, and one of the problems of organized medicine is, how can we provide incentives that these young men may grow and develop in the profession.

These are just some of the problems which the advance of our profession in a changing world has brought. Two years ago, at Banff, the C.M.A. appointed a committee to study the subject and make recommendations for a solution of some difficulties. After a careful study, that committee reported to the Association at Winnipeg last year and advised the establishment of a College of General Practice within Canadian organized Medicine, with the following aims and objects:

1. To establish an academic body with broad educational aims.
2. To arrange for undergraduate teaching by and for General Practitioners.
3. To arrange for the presentation of postgraduate education for general practitioners.
4. To arrange for research in general practice.
5. To arrange for publication of original articles by general practitioners.
6. To arrange for hospital staff appointments for general practitioners.
7. To provide suitable recognition to members in the field of general practice.
8. To do all things necessary to maintain a high standard in general practice.

The report with its recommendation was adopted and an organizing committee was appointed to proceed at once with the establishment of the proposed College. The work has been going forward as indicated by a brief report by Dr. Glenn Sawyer, secretary of the founding committee in the December number of the Journal. It is expected that the College will be ready by March to receive applications for membership. The College will be officially started on its career at the June Meeting of the C.M.A. in Vancouver.

It was early recognized that the organization must have a full time secretary if it is to succeed. An outstanding man has been secured and will begin his work in March.

The item of finance is causing some anxiety. The cost will be about $25,000 per year. The first two years will be the most difficult until the College gets firmly established. For this reason, a Foundation Fund has been set up and the success of the venture will largely depend upon our financial response to the appeal. One can hardly doubt that there are many who will gladly subscribe $50 or $100 to this project which can and will mean so much to our profession.

This is a new venture in Canadian Medicine. It was stated at the outset that the establishment of the Royal College of Physicians and Surgeons was a great forward step. This is a second and complementary one. It is intended more particularly for the younger members of the profession. It points to the future. It reaffirms our belief that the family doctor *is* the backbone of medicine and that backbone must grow straight and strong and not shrink and twist with the years. Let the younger men support the venture enthusiastically. Theirs is the future. As for us older ones, let us not ask "What is there in it for us?" Rather, let us see our opportunity. Years ago our profession did us the honour of numbering us among its members. What have we done in return for that great honour? Here is our opportunity. Let us support with our interest and our money the College of General Practice of Canada.

LE COLLEGE DE PRATIQUE DE LA MEDECINE GENERALE

CHAS. L. GASS, M.D., *Tatamagouche, N.S.*

L'ESSOR SOUTENU et accéléré des connaissances humaines a causé l'avènement nécessaire du spécialisme. Ces changements sont surtout évidents en médecine, comme le savent fort bien les membres de la profession.

Une des grandes étapes dans le progrès de la médecine canadienne des vingt dernières années fut l'établissement du Collège Royal des Médecins et Chirurgiens pour la formation de spécialistes compétents et l'octroi de diplômes dans les différentes branches de la profession. Ce progrès ne s'accomplit pas sans réaction contraire. Il faut du temps pour découvrir et supprimer certaines faiblesses. Nous ne nous occupons pas ici, quoique nous devrions peut-être le faire, des effets néfastes sur le spécialiste moderne qui sacrifie l'envergure à la profondeur: sur certains rapports, du moins, la comparaison avec ses ainés qui arrivaient au spécialisme en passant par la médecine générale, lui est défavorable. Mais l'Association Médicale Canadienne s'est préoccupée depuis quelques années de l'effet qu'a le spécialisme croissant sur l'état d'esprit et la condition de l'omnipraticien.

On entend souvent dire que personne ne peut suivre de près tous les progrès de la médecine. Le médecin zélé et laborieux a réussi à se tenir au courant, avec un succès remarquable. Il s'est perfectionné tant en envergure qu'en profondeur dans les dernières trois ou quatre décades, et cette réussite n'a pas reçu toute la reconnaissance qu'elle mérite. L'accroissement du savoir et de l'efficacité n'a pas été le monopole des spécialistes de la profession. Cependant on semble croire dans certains milieux que l'omnipraticien perd de son prestige tant au point-de-vue social que professionnel. Un médecin de l'ouest, reconnu pour son ardeur et son énergie, a mis les points sur les i en disant qu'il en avait soupé de s'entendre appeler le clef de voûte de la profession alors qu'on le traitait comme un vulgaire pavé. Le bien fondé de ces opinions n'est pas facile à évaluer, il n'en est pas moins sûr qu'en certains lieux, l'omnipraticien est en butte à des difficultés qui le gênent dans l'accomplissement de sa mission, tout particulièrement en ce qui concerne ses rapports avec l'enseignement et les hôpitaux. Une des raisons de ceci, est l'absence de reconnaissance officielle de la qualité de ceux qui dans la pratique de la médecine générale

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ont su par le travail et les études post-universitaires, se maintenir à la page. Aucun certificat d'excellence n'existe en médecine générale qui puisse se comparer à ceux que le Collège Royal des Médecins et Chirurgiens décerne comme recommandation à ses spécialistes reconnus. Quoiqu'on pense de la vraie valeur de ces parchemins, on doit reconnaitre que de nos jours, ils sont tenus comme des plus désirables et même nécessaires en maintes circonstances. Plusieurs omnipraticiens méritent certainement quelque marque de distinction.

L'omnipraticien—le médecin de famille—est-il en voie de perdre son rang dans la société? Nous vivons dans un âge qui est non seulement matérialiste mais aussi incrédule. Peut-être l'omnipraticien ne jouit-il plus du don d'omniscience que l'on prêtait à ses prédécesseurs. Peut-être son auréole devient-elle diaphane, mais il ne aut pas pour tout cela en blâmer le développement du spécialisme. Ce que la population demande presqu'à genoux, c'est un plus grand nombre de docteurs de famille, bien entrainés, progressifs, probres, et non pas plus de spécialistes. Je crois que la demande de spécialistes provient, comme se doit, des omnipraticiens qui ont besoin de leur secours et qui savent l'apprécier. L'omnipraticien doit se rendre à l'évidence que la sécurité de son état dépend de lui-même.

Il y a eu un grand engouement pour le spécialisme depuis la guerre. Le spécialiste a remplacé le médecin de famille dans l'idéal de plusieurs étudiants en médecine des plus sérieux. Dans l'après-guerre immédiat, les facilités pour l'entrainement de spécialistes ont été exploitées à la limite. On accuse quelquefois les écoles de médecine et les hôpitaux affiliés à la faculté d'être, sous l'influence et le zèle du Collège Royal des Médecins et Chirurgiens, plus intéressés à produire des spécialistes que des omnipraticiens. Ce n'est qu'au détriment de la profession qu'un tel état de chose puisse exister. La médecine canadienne se porterait certainement mieux si l'omnipraticien pouvait exercer d'avantage l'influence qu'il possède et le zèle qui l'anime. Il est manifeste que l'élan vers le spécialisme s'est déja ralenti. Un plus grand nombre de jeunes médecins s'orientent vers la pratique de la médecine générale, et l'un des problèmes de l'organisation médicale est de savoir comment stimuler chez ces jeunes gens le désir de s'élever et de se perfectionner dans l'exercice de la profession.

Ceux-ci ne sont que quelques uns des problèmes posés par le progrès de notre profession dans un monde en évolution. Il y a deux ans à Banff, l'A.M.C. a nommé un comité chargé d'étudier le sujet et de fournir des recommandations pour en résoudre les difficultés. Après une étude approfondie, le comité a fait rapport à l'Association l'an dernier à Winnipeg, et a recommandé l'établissement d'un Collège de la Pratique de la Médecine Générale au sein de l'organisation médicale canadienne, dont les buts seraient comme suit:

1. Etablir un corps enseignant avec d'amples visées éducatives.
2. Organiser l'enseignement universitaire pour former des omnipraticiens par des omnipraticiens.
3. Organiser un enseignement post-universitaire à la portée des omnipraticiens.
4. Organiser un programme de recherche en pratique de la médecine générale.
5. Organiser la publication de travaux originaux par les omnipraticiens.
6. Voir à la nomination d'omnipraticiens dans le choix du personnel des hôpitaux.
7. Assurer la reconnaissance officielle des membres dans la pratique de la médecine générale.
8. Faire tout en son pouvoir pour conserver un niveau élevé d'intégrité dans la pratique de la médecine générale.

Le rapport et ses recommandations furent acceptés et l'on nomma un comité d'organisation pour procéder sans délais à l'établissement du Collège proposé. Le travail avance comme l'indiquait un bref compte-rendu du Dr. Glenn Sawyer, secrétaire du comité de fondation, publié dans le numéro de décembre du Journal. Le Collège devrait être en mesure de recevoir des demandes d'adhésion dès le mois de mars. Il sera lancé officiellement à Vancouver en juin lors du congrès de l'A.M.C.

On s'est tôt aperçu que l'organisation doit avoir secrétaire à temps complet si elle compte réussir. Nous avons obtenu les services d'un homme remarquable; il doit se mettre à la tâche en mars. La question financière est une source d'inquiétude. Le coût sera d'environ $25,000.00 par année. Les deux premières années seront les plus difficiles jusqu'à ce que le Collège soit fermement établi. A cette fin, un Fonds de la Fondation a été ouvert et le succès de cette entreprise dépendra en une large mesure de nos contributions. Il est certain que plusieurs n'hésiteront pas à contribuer des sommes de $50 ou $100 à ce projet si important à notre profession.

Cette entreprise est une innovation en médecine canadienne. On a affirmé lors de la fondation du Collège Royal des Médecins et Chirurgiens qu'il était une étape importante. En voici une seconde qui lui fait pendant. On la destine surtout aux cadets de la profession. Elle trace la voie de l'avenir et réitère notre conviction que le médecin de famille est la clef de voûte de la médecine et qu'une clef de voûte doit être forte et solide, et ne doit pas s'effriter avec les années. Que les jeunes appuient cette entreprise avec enthousiasme. L'avenir leur appartient. Pour nous, les ainés, ne cherchons pas à voir ce que nous pouvons en retirer. Qu'elle représente plutôt l'occasion de rendre à notre profession l'honneur quelle nous fit jadis de nous accepter parmi ses membres. Encourageons donc par notre intérêt et nos finances le Collège de Pratique de la Médecine Générale du Canada.

COLLEGE OF GENERAL PRACTICE OF CANADA

Provisional Executive Committee.—(1) President, Dr. M. R. Stalker, Ormstown, Quebec; (2) President-Elect, Dr. J. H. Black, Vancouver, B.C.; (3) Honorary Treasurer, Dr. Glenn I. Sawyer, Toronto, Ont.; (4) Chairman of Board of Representatives, Dr. C. L. Gass, Tatamagouche, N.S.

Additional members: Dr. Armand Rioux, Quebec, P.Q.; Dr. J. Wendell MacLeod, Saskatoon, Sask.

Executive Director.—Dr. W. V. Johnston, Lucknow, Ont. (To assume office March 1, 1954).

Provisional Board of Representatives.—The Executive Committee plus the following representatives of the Provinces: British Columbia, Dr. E. C. McCoy, Vancouver; Alberta, Dr. Pat Rose, Edmonton; Saskatchewan, Dr. Fritz Werthenbach, Unity; Manitoba, Dr. Jack McKenty, Winnipeg; Ontario, Dr. William Wilford, Wiarton, Dr. Maurice Hobbs, Millbrook; Quebec, Dr. Armand Rioux, Quebec, Dr. Hans Geggie, Wakefield; New Brunswick, Dr. Melville Rice, Campbellton; Nova Scotia, Dr. Alex MacLeod, Dartmouth; Prince Edward Island, Dr. L. G. Dewar, O'Leary; Newfoundland, Dr. John Walsh, Manuels.

COLLEGE OF GENERAL PRACTICE OF CANADA

APPLICATION FOR MEMBERSHIP

(PLEASE PRINT OR TYPE)

Name in full.....Date.....

Office address.....City.....Prov.....

Residence address.....City.....Prov.....

Place and date of birth.....

Medical Education—Medical school.....

—Date of graduation.....Degree.....

Licensed to practise in.....province(s)

Other qualifications and diplomas.....

.....

.....

Internships—Hospitals.....From.....To.....

.....From.....To.....

.....From.....To.....

.....From.....To.....

Teaching appointments.....

How long have you been engaged in the active general practice of medicine?.....

Are you now so engaged?.....

How long did you serve as an assistant to a general practitioner?.....

With whom did you so serve?.....

From.....To.....

Are you a member of a hospital staff(s)?.....

In what category?—Honorary ☐ Active ☐ Associate ☐ Consulting ☐ Courtesy ☐

Name of hospital(s).....

.....

Member of.....local medical society

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Educational Activities in Past Two Years:

1. Scientific meetings attended: *Approx. No. of hours*

County medical society meetings:

Conventions—Provincial Association, C.M.A. and others (specify and give dates):

---

2. Post-graduate courses taken (specify—giving dates):

---

3. A. Hospital staff meetings:

B. Other medical activities, such as hospital rounds, medical papers, submitted or published, planned reading courses, book reviews, case history reports submitted for publication, community health services.

In applying for membership in the College of General Practice of Canada, I certify that I am a member of my Provincial Medical Association and the Canadian Medical Association or L'Association des Médecins de Langue Française du Canada.

I am enclosing a cheque for my fees for 12 months in the class of Active ☐ Associate ☐ membership.

I understand that the money will be refunded if my application is not approved. In submitting this app lication, I hereby agree to abide by the Regulations of the College of General Practice of Canada.

References: Name Address

1. ---

2. ---

Signature of Applicant

Recommended for membership—Honorary ☐ Active ☐ Associate ☐

Appointment not recommended ☐ Appointment deferred ☐

Remarks:

Provincial Credentials Committee

Membership applications should be sent to:
The College of General Practice of Canada,
244 St. George Street,
Toronto 5, Ontario.

Dr. W. V. Johnston, of Lucknow, Ont., has been appointed Executive Director of the College of General Practice.

Dr. W. V. Johnston

This appointment calls for unusual qualities of administrative experience, of patience, of determination, of clear thinking; it is a *sine qua non* that the new Director should have been a general practitioner of long experience.

Dr. Johnston has amply demonstrated by his work on both national and provincial levels that he has these qualities. He takes up his new and arduous duties with the best wishes of his many friends for the success he deserves and should achieve.

MISCELLANY

PRESIDENTIAL ADDRESS*

J. W. REID, M.D., *Halifax, N.S.*

[*We reprint with pleasure Dr. W. Reid's presidential address to the Medical Society of Nova Scotia on the occasion of its Centenary meeting in Halifax, on October 9, 1953. Dr. Reid employs a delightful humour as he passes his keenly discerning eye over medical history.*—EDITOR.]

It has been the custom for some years past for the president at the annual dinner to speak pertinently of matters urgently concerning the medical society, to give an account of his stewardship, and end by thanking his officers and committees. Tonight I will change that order and begin by thanking the officers and committees first. This has been a heavy year and one of its greatest surprises and pleasures has been the willingness and vigor with which the committees of this society took hold of the various and heavy burdens which were imposed on them during the past year. . . . It is no wonder that Nova Scotians are so famous abroad when Nova Scotians at home are so competent!

When on May 7, 1853, the Halifax Medical Society met to consider the unjust treatment of medical bills recently before the House, they decided to do what Halifax doctors have always done when in trouble—retire to their chambers, bare their teeth and call upon the doctors of the province to come to their rescue. Thus it was that letters were sent to ninety-odd duly licenced doctors in the province, and over seventy replies favourable to a Provincial Union came back. Thus out of the need for strength was our Society born! There our story begins!

Now, gentlemen, I realize that it is my duty to cover in detail the events of the last hundred years! What an interesting task it would be! But fortunately for you, it is impossible. It seems reasonable, however, to take a few minutes to make some comparisons of the circumstances of medical practice then and now. The first is that poor unfortunate fellow who is as constant and close to the doctor as his shadow, the patient! One hundred years ago life was simpler and harder for everyone, and much more so for the sick. The infectious diseases were largely uncontrolled, there was no anæsthesia, no asepsis, and deaths from tuberculosis, diphtheria, puerperal sepsis, and typhoid fever were staggering. There were no hospitals, no laboratories, no x-rays and practically no specifics. Ah, you city-bred weaklings, how would you like to have amputated a leg before the days of anæsthesia or practised midwifery in those dark and pain wracked days before natural childbirth? We look back at the hardships of travel, the absent diagnostic aids, the limited drugs and the loneliness of the practitioner and we wonder how they had the courage to carry on, and why, when they had seemingly so little to offer, the patient revered them so. We in this time can never fully appreciate or understand the respect and affection with which the sick regarded the physician of a century ago, who was called not because of the wonder drugs at his disposal, or the marvellous and certain cure he would work, but because he was kind and understanding, and knowing at least more of sickness than they, could be relied upon to do his very best in their time of trouble! Alas! As we came to know disease a little better, we came to know the patient less.

In those days there was no place to be sick but home! And a fairly efficient hospital it was! There was often a spacious airy sick-room with sunshine, good food and loving, if untrained, nursing care. There were servants to cook and clean and maiden aunts to wait upon and spoil the patient. There were the spacious double parlours for restful convalescence and lawns and gardens for quiet exercise and fresh air.

How all this has changed! The spacious bedroom has become a closet with double bunks, the servants are in the retail trade, the maiden aunts are in legitimate business, the double parlour has become the double garage, and the garden is in a paper box from the florists! Is it any wonder then, that as you pull your chair up in the hall and reach into the bedroom to feel his pulse, that he responds, when you ask to see his tongue, by showing you his Blue Cross Card instead? Here is no docile, adoring patient, but one who sits up, if he has the strength, and demands, "Cure me Doc, or else." The patient has changed! And I make this comparison because, regardless of accusations of lay groups, all the blame for the high cost of sickness does not lie with the medical profession! The patient himself must bear no small measure of the blame. How did this change occur? Who can tell? Perhaps when the doctor turned from the gentle humility of great ignorance to the brusque arrogance of a little learning, the patient, that chameleon,

*Slightly abridged. By kind permission of the *Nova Scotia Medical Bulletin.*

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changed with him! He listened, read and observed. He became more enlightened and more cynical until today, he has lost faith in his mother's homely nostrums, has lost confidence in himself and his family doctor, is fast losing his faith in God, and has already lost many of the comforts of his religion, so that with every slight disturbance of body or mind, he turns to the nearest specialist to protect and prolong the only life he knows or feels sure of. Such a one soon develops the traits of the addict so that every taste he gets of free health service arouses in him an appetite, which if appeased, may go on to an insatiable hunger for medical care.

That is the ominous portent for those upon whose shoulders rests the burden of health planning in this country, and we who know best the facilities and capabilities of the profession and the desires and demands of the patients, beg that those who carry this responsibility, walk into the wilderness warily, and slowly!

And what of the doctor? How strongly must a man have been called one hundred years ago to enter the medical profession! How anxious he must have been to serve in humble capacity the suffering people around him. How discouraging it must have been to sit helpless at the bedside of children dying of diphtheria, and what nerve it must have taken to do the surgery of that day. Such a man must have been as courageous as a lion, as tender as a mother, as swift as an adder, and as gentle as a maiden in love! Where could their like be found today—save in Cape Breton!

In those days the doctor was everything; physician, surgeon, midwife, apothecary, confidant and friend. He worked largely alone without diagnostic aid, without consultants and with a meagre list of specific drugs. He travelled almost entirely on horseback, and his work was done in his office and in the homes. There was in all Nova Scotia, no hospital except the old Bridewell in Halifax, and that without any facilities. Yet, so great was his prestige with the public, that when he fell ill himself, heavy traffic would avoid a street in which he lay, straw would be strewn on the roadway to deaden the sounds of wheels and passers-by converse in quiet tones.

Picture the weary round of house calls, the hours of waiting in attendance on obstetrical cases in the home, the busy office, the clanging night bell and the dreary journey its summons must often have demanded, and you see a man of great physical and spiritual stamina, his life endurable only in the conviction that those who went before him had it worse and bore it better.

What has become of that courageous figure today? The long beard shorn by the development of asepsis gave way to the side whiskers, and these in turn to the moustaches and finally when all the hair was shaved away, we find not one but many faces! The stalwart figure of that other time is broken into many bits and the sum of all the parts seems less than the whole.

One of the brightest faces to emerge is that of the public health worker! If we look back to the years before the formation of the Nova Scotia Medical Society in 1853, before the formation of the Halifax Medical Society in 1844—if we look back to the very beginning of civilization in Nova Scotia, we find a continuous recurrence of epidemic disease beginning with the scurvy which killed 50% of Champlain's garrison in 1605. When in 1606 he attributed the halving of his death rate to better living conditions, he recorded the first public health study in the new world; and the post mortem which Deschamps performed on one of the victims was probably the first autopsy in America. Smallpox raged among the Indians in 1694 and again in 1749, and just a few months after the founding of Halifax, it killed a thousand of the settlers. Again in 1801 there were 8,000 cases of smallpox recorded in and around Halifax.

Typhus and smallpox together caused 800 deaths out of a population of 11,000 inhabitants in Halifax in 1827. Is it any wonder that the thoughts of medical men and legislators turned to methods of prevention—and the first of these was the quarantine of infected ships and the appointment of a Port Physician in 1761. This was followed in 1779 by the appointment by the Governor of health officers in all counties and districts with wide powers to enforce the quarantine and health regulation then in force—the burning of infected clothing and bedding, burials, etc.

In 1832 the first Central Health Board was formed and this remained until 1870. The first mental hospital was built about 1854 and the first general hospital in 1859. The first organized attack on tuberculosis was begun in 1894. The first Department of Health was organized in 1904 and the first Minister in 1931.

The diagnostic and treatment services were begun in the 1920's, expanded in the 1930's and in some phases, mark these dreadful words, made free and compulsory in the 1940's. Ah well! So the times change and free men, dulled by the sweet wine of Socialism, do not yet feel the chains being forged to their lives. Now we hear of new and broader ventures in the offing. The Nova Scotia Medical Society is delighted by this evidence of growth and vigour and we assure our public health colleagues that as they struggle to emerge from the stench of their cess pools and sewers, members of our Society will always be on hand to lift them out of their dank world into the more soul-satisfying atmosphere of the healing art.

Consider the Radiologist: one hundred years ago equipment for visualizing the hidden structures of the body was simpler and more portable than it is today. It consisted of the eye, the ear, the fingers and the brain of the examining physician. But now the x-ray has replaced all these—not excluding the brain. When the German physicist Conrad Roentgen discovered the x-ray in November 1895, he withheld from the world his report until mid January 1896. This set the pattern for delayed x-ray reports which has been the vogue ever since!

When about the turn of the century a young graduate of Toronto University, Herbert Weaver, came to Halifax enthused with the idea of x-ray as a specialty, he found a complete absence of equipment. Presently he was commissioned to go to Boston and obtain equipment for installation in the Victoria General Hospital, and the department began to function about 1903 under his direction. The apparatus was primitive, though deadly, there was but little technique and no literature. It was their proud delight for many years to demonstrate to skeptical observers the solid outline of a silver dollar between the covers of a thick family bible—if you had a silver dollar!

Today, there is no phase of medicine or surgery which could function successfully without the diagnostic aid and treatment facilities of this important branch of medicine, which in these past fifty years has developed a strong society, a comprehensive literature, elaborate technique and more powerful and deadly apparatus.

Now, with commendable enthusiasm, they claim to be able to visualize your soul through a pile of greenbacks—if you have the greenbacks!

Consider the Anæsthesiologist: seven years before the founding of the Nova Scotia Medical Society, a Boston dentist, Dr. Morton, used sulphuric ether as an anæsthetic in dental surgery. It was the year 1846. The patient had hardly recovered from the anæsthetic before a Halifax dentist, Dr. Van Buskirk arrived in Boston to learn the technique. The dentist was hardly back in Halifax before a prominent Halifax surgeon, Dr. D. M. Parker waited on him and requested that he administer to him an anæsthetic. The surgeon, having made first-hand acquaintance with the new method, had the ether administered the next day for an amputation of a leg, the first recorded use of general anæsthesia in Nova Scotia.

About this time, Sir James Simpson in Edinburgh, was experimenting with all manner of drugs to produce painless sleep, and finally administered to himself in 1847, an inhalation of chloroform. Soon he was in a profound sleep from which he fortunately recovered to tell the world of his discovery.

Since then, innumerable new techniques and substances have been used and today we find that the anæsthesiologist who began as a general practitioner pouring ether out of a bottle, is currently pouring the anæsthetized G.P. back into the bottle for storage on a museum shelf. Alas, the specialty that was conceived in the noble hope of making surgery painless for the patient has matured to the lowly task of making operations safe and easy for the surgeon!

What of the surgeons? No field of human endeavour has made greater progress either in technical skill or in benefits to the comfort and longevity of the community than have these grisly knights of the mask and scalpel. At the beginning of the century of medicine which we are celebrating tonight, one of the professors of surgery in Edinburgh was wont to say that a surgeon who attempted to open the abdomen should be indicted for murder. Yet, within a quarter century of the utterance of these words, there was brought to Nova Scotia from that same city, the technique which Lister had devised and which John Stewart brought so promptly to his native province—that technique which has made possible the exploration of every cavity of the body and set the bounds of successful surgery at the utmost limits of biological tolerance. Today these men can hold your throbbing heart within their hands and none would mark them save for honour. So has the scene changed that those who a century ago dragged their patients through their operations asleep, but with their incisions infected and dirty, pass them quickly on today dazed but clean—completely cleaned!

What of the future of surgery? After our session on fees this afternoon, I say "leave them to heaven!" For now that Maritime Medical Care is affiliated with Trans Canada Medical Services, the restless minds whose urge it is to conceive these vast expansions, will form an Interplanetory Medical Service Unlimited! Then will the surgeon truly come into his own. He can raise his fees as high as the stars, spend his days sailing through the tranquil corridors of space and live forever in celestial grandeur.

Think for a moment of Psychiatry: From the time in the late eighteenth century when the French humanitarian, Phillip Pinel, struck the chains from the lunatics of Paris, the care of the mentally ill has been a story of steady but slow progress. How slow is indicated by the fact that more than a quarter century after Pinel's death in 1826, Nova Scotia had no hospital facilities whatsoever for the custodial or other care of the mentally ill. So pressing in fact, was the need that in 1844 the then mayor of Halifax, Hon. Hugh Bell, offered to donate his year's salary of three hundred pounds toward the building of a lunatic asylum. (Obviously the then mayor was a little suspicious of some of his aldermen too)! The medical men meeting to consider this and other matters presented by the mayor, formed themselves on October 26, 1844 into the Halifax Medical Society. The mayor subsequently agreed to let his offer stand towards the construction of a general hospital *if provision was made in it for the care of lunatics*. This battle is still being waged a century later though that hospital was never built due to lack of public interest. It was thirteen years later, after the intervention of the famous Dorothea Dix, that the Nova Scotia Hospital was built in 1857, and then called Mount Hope.

From that time psychiatrists remained in hiding, their own time biding, until they had grown great enough in numbers, strength and learning to break out (they prefer the term emerge) from behind the walls of these institutions for the care of the mentally ill, into the free society. This break out, beginning as it did about a quarter century ago, soon assumed the proportions, though unfortunately not the direction, of a migration of the leemings. So powerful was the impact of these colleagues upon the community of health care that the old guard physicians are still stunned and the psychiatrists themselves astonished!

Now we know that the time has not yet come when there is a couch under every patient, nor a psychiatrist by every bed, but all over the world there is a constantly increasing awareness of mental health problems and of their significance in the etiology of many organic diseases, which has led to a steady encroachment of psychiatry on the field of practice. The time has come when we must accurately determine whether the incidence of mental illness in minor and major forms is truly great enough, as the psychiatrists claim, to justify their demands for more and more teaching hours in the medical curriculum and ever expanding facilities and personnel. If such as they claim be so, then we must do all in our power to facilitate their emergence from behind their mahogany desks and tape recorders and get them out into the homes and sickrooms where they can do the most good!

Finally, let us compare for a moment the educational opportunities which were available to young men then and now.

In those years there was no medical school in Nova Scotia and only a fraction more than half the medical men in practice at that time had proper medical degrees. Of the 114 men practising in Nova Scotia in 1853, only sixty had medical degrees and half of those were Edinburgh. Licensure at that time was by a committee of medical men appointed by the government, and the examination was generally held in one or other doctor's office and was entirely oral.

The American schools of this time were considered inferior to those of Britain and the Continent, plagued by innumerable diploma mills which were entirely dishonest and irrepressible and which continued to flourish even into the twentieth century. In the better American schools, even as late as 1870, the course consisted of only two years of five months each, with low matriculation standards. No attendance records were kept and hospital and clinical teaching was often inadequate. One of the common ways of obtaining medical education in the early days was by apprenticeship to a man in practice, a method being reconsidered today.

At the time of the founding of the Halifax Medical College in 1868, the course was of three years' duration and compared favourably with the better American schools, having been patterned largely after the Edinburgh school where the course was also at that time three years. From that day to this as new knowledge was gained, new courses have been added, always by a process of accretion, adding years to the course and months to the years until the present seven years of nine months each from high school to basic qualification. And currently the American schools are increasing to eight years and expecting us to do the same. In addition, young men who wish to prepare for certification in a specialty, must spend an additional two to four years in resident training! Twelve years from high school to his first earning—one quarter or more of his productive lifetime in costly education!

What is the future of medical practice and medical education? Can the medical schools anticipate the trend and train the youth to meet the changed conditions, or must they, like the politicians, follow after the mob because they are their leaders. There is a challenge to medical educators today—a challenge which all are fearful to accept—the challenge of complete and drastic revision of the medical curriculum!

In the light of the truth that a sound and progressive medical service must always rest upon an educated profession, medical teachers have been afraid to drop a line or a lecture from the curriculum lest they be accused of lowering the standard of medical education. The result is an overcrowded course and a driven student!

Education, however, is not to be found in a harried

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brain overcrowded with facts and the knowledge of technical skills, but rather in a mind trained to recognize pertinent facts, to consider them rationally and to act upon them wisely. Man cannot reason without facts, nor can he think with facts alone! One is reminded of that verse of the lover of leisure who wrote:

"Oh may we not be in such desperate haste
That we may not one hour from the day's round waste,
And in some quiet talk with fellow men
Have strength renewed and minds refreshed again."

It has been said that medical education today is not adequate to or compatible with the safe application of the dangerous techniques which science has placed at our command, and that the universities and medical schools have been guilty of favouring their youngest child, science, almost to the exclusion of the older cultural subjects. One is reminded of a verse from one of Eugene Field's childhood poems which reads:

"Oh mother, my love, if you'll take my hand
And go where I ask you to wander,
I'll lead you away to a beautiful land,
That dreamland that's waiting out yonder!"

It seems to many that the sciences may be leading the universities and the medical schools down a primrose path to a dreamland where truth is never found and where faith, its only substitute, may be lost forever. Truly, medical training must continue to be based upon the scientific method, but the depth of culture in the stream of medical education must not give way entirely to the bright and fascinating rapids of science—lest the tender care of the sick be wrecked on the hard, unsympathetic rocks of research!

We who profess some concern for the availability of medical services must have the vision and courage to plan the training which we believe will best enable the medical schools to provide in quality and numbers the practitioners of tomorrow. Modern civilization would not be possible had not men found a way to make better things, in less time, at less cost. We too can find a way!

Though we may work in vastly different fields, though we may have differing opinions on education and practice, each and every one of us is aiming at the self-same goal—to succour the sick and banish disease from the earth! To that end this Society and the Canadian Association has an opportunity, nay, an obligation to develop the best medical service in the world! Poised as we are at the meeting place of the vast medical culture and experience of Europe and America we can and must draw the best from each! It is my belief that if governments will long enough resist the pressures of socialism; if they will consult with, be directed and restrained by the medical profession, we can, in this province and in this nation, develop, within the pattern of private enterprise and the dignity of personal responsibility, a medical service which will be the comfort of the sick, the pride of the profession and the envy of the world.

In conclusion may I borrow from the words of the Psalmist and say—"Surely God has placed the feet of the medical profession in a large room." Let us endeavour always to be worthy of that magnitude!

RE-SURVEY OF PHYSICIANS IN CANADA

March, 1954, by the Department of National Health and Welfare

The Physicians Register of the Department of National Health and Welfare in Ottawa plans to conduct a re-survey of physicians in Canada beginning the latter part of the month of March. The Canadian Medical Association hopes that all physicians will co-operate with the Department by participating in the survey.

The original records of the Physicians Register were set up by the Canadian Medical Procurement and Assignment Board during World War II. When that body was dissolved at the end of the war, the Canadian Medical Association was anxious that the records be maintained but, because of financial considerations, was unable to undertake the responsibility itself. When the Department of National Health and Welfare was approached in the matter, the Canadian Medical Association encouraged the Department to accept.

The periodic statistics based on the records of the Physicians Register, published in the series of reports "Survey of Physicians in Canada" by the Department of National Health and Welfare, have been very useful to the Canadian Medical Association as well as to other medical bodies in Canada. Maintenance of the Register is possible to a great extent from indirect source material, such as the registers of the Provincial Colleges of Physicians and Surgeons, but periodic re-surveys are also necessary since not all the information tabulated can be obtained from these indirect sources.

Many physicians will be familiar with the type of postcard questionnaire sent out by the Physicians Register when a change in location is noted, although, in anticipation of the complete re-survey, these have not been used extensively during the past year or so. The form is easy to fill in and is then merely dropped in the mail. The present form is expanded slightly over that usually used so that additional special studies may be made for the group as a whole.

Physicians who do not receive a questionnaire form by April 30 at the latest should write immediately to the Physicians Register of the Department of National Health and Welfare, Ottawa, requesting a questionnaire and stating clearly the address to which it is to be forwarded. As a further aid to ensuring completeness in the re-survey, a supply of blank questionnaires will be forwarded to the larger hospitals. In particular, interns, whether junior or senior, physicians doing postgraduate study, and physicians who have recently come to Canada from other countries are requested to enquire about obtaining questionnaires at that source.

FILL IN YOUR QUESTIONNAIRES AND MAIL THEM PROMPTLY, PLEASE!

Association Notes

TRAVEL ARRANGEMENTS TO VANCOUVER

RAIL TRAVEL

The Canadian Passenger Association has authorized special convention rates for members of the Canadian Medical Association and their families travelling by rail to the Annual Meeting in Vanvouver. Identification certificates permitting members to purchase tickets at a considerable saving may be obtained on application to the General Secretary, Canadian Medical Association, 244 St. George Street, Toronto 5, Ontario.

Dates of Starting Journay:

From stations in Western Canada, west of Armstrong and Fort William, June 7 to 16.

From stations in Eastern Canada, east of Armstrong and Fort William but not including Newfoundland, June 4 to 13.

From stations in Newfoundland, June 1 to 10.

Fares (adult):

Going and returning same route—one and one-half times the adult normal one-way first, intermediate or coach class fare applying via route used as shown in tariffs plus 25 cents.

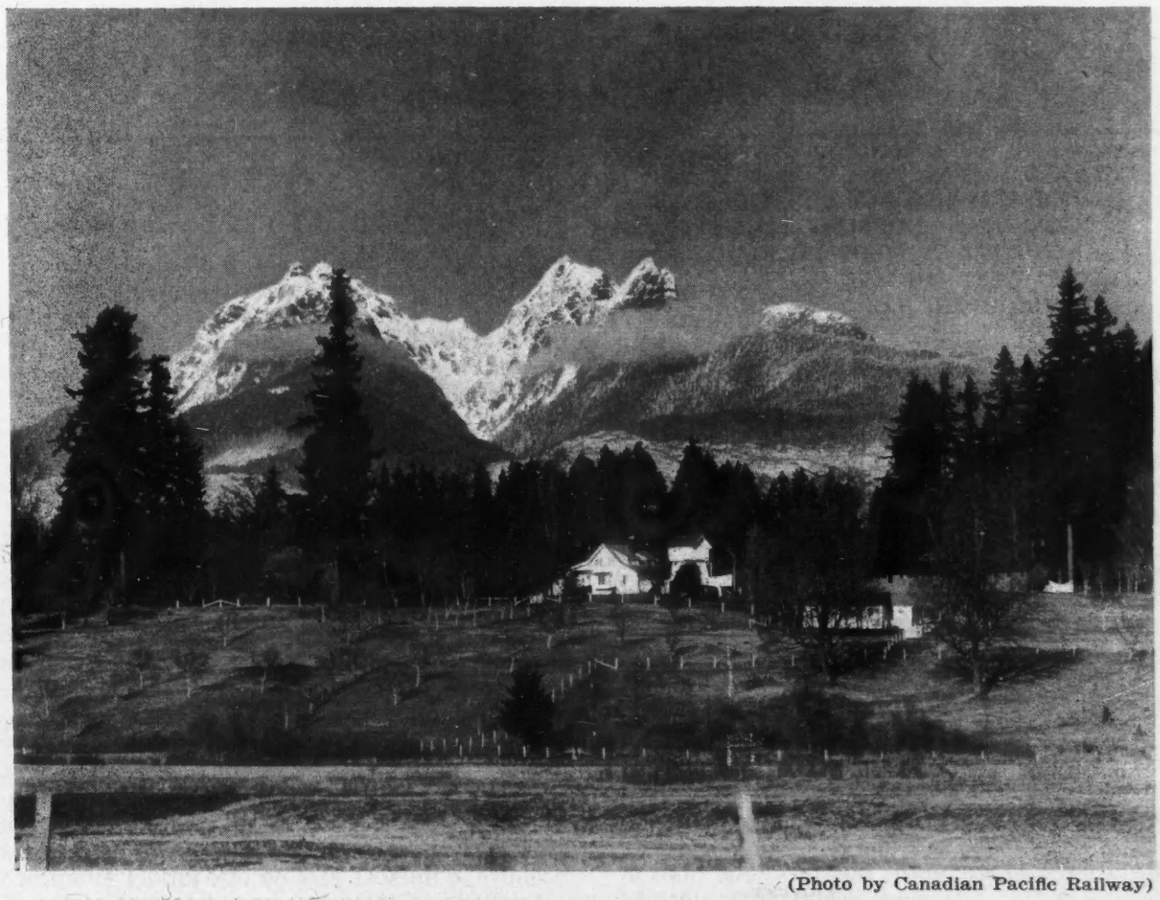
Diverse routes.—Three quarters of the one-way first, intermediate or coach class fare, as the case may be, applying from starting point to destination via route travelled on going trip plus three quarters of the one-way fare of the same class applying from starting point to destination via route travelled on return trip plus 25 cents.

Return Limit:

Thirty days after the date on which the ticket is valid to start the going journey. Return trip may commence on any date within the final return limit, and passenger must reach original starting point not later than midnight of final return limit.

AIR TRAVEL

The Identification Certificate Plan does not apply to travel by air. Trans-Canada Air Lines has, however, indicated that three types of service will be in operation to Vancouver:



(Photo by Canadian Pacific Railway)

Farm in lower Fraser valley, B.C. Golden Ears Mt.

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RETURN FARES TO VANCOUVER (BY AIR)

A. Regular	—from	Halifax	$401.10,	Montreal	$326.50,	Toronto	$288.55,	Winnipeg $161.90
B. Tourist	— "	"	365.00,	"	290.00,	"	256.00,	" 144.00
C. Group	— "	"	356.55,	"	290.25,	"	256.50,	" 143.90

NOTE:—The Group rate applies when an organized group of ten or more persons travel together on the going portion of the journey and relates only to Regular service.

It is recommended that members attending the Annual Meeting in Vancouver make their travelling reservations at an early date.

COLOUR TELEVISION AT THE ANNUAL MEETING

Many members will recall with pleasure the first Canadian showing of medical colour television which took place at the Annual Meeting in Montreal in 1951. Again sponsored by Messrs. Smith, Kline and French, Montreal, a full scale program of varied clinical topics will be presented this year at the Annual Meeting in Vancouver.

Remarkable technical advances have been made in the few short years since this medium was first applied to the dissemination of medical information and this year the presence of a projection screen will permit five hundred viewers to see the same colour image without distortion.

The screens to be used are 5 x 6 ft. They provide a picture 27 times larger than the 12" receivers previously used.

Under the chairmanship of Dr. J. F. McCreary, an active committee of our Vancouver colleagues has been preparing a program with features of interest to every practitioner. It will be noted that the scientific meeting at Vancouver starts on Monday, June 14 and extends through Friday, June 18. The first half is presented in colour television and the remainder by an outstanding assembly of speakers.

The Association is indebted to Messrs. Smith, Kline and French, Montreal, for making this colour telecast available and it is our privilege to present the following details of the projected attractions.

"The Pacific Shore in '54"

C.M.A. CONVENTION - JUNE 14 - 18, 1954

Make Your Reservation NOW

MAIL THIS APPLICATION

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CHAIRMAN—HOUSING COMMITTEE,
ACADEMY OF MEDICINE BLDG.,
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MOTEL — Single ☐ Suite ☐ Kitchen facilities ☐

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Date of arrival ..... A.M.— P.M.— Departure .....

Are you driving your own car? .....

Do you wish a U Drive car reserved? .....

Rooms will be occupied by .....

Name	Street address	City	Province

(attach additional names if necessary)

Send Confirmation to Doctor: .....
(please print)

Street .....
City .....
Province .....

PRELIMINARY PROGRAM OF COLOUR TELEVISION

Monday, June 14, 1954—9.30-12.10 p.m.

Radical Mastectomy—Demonstrator to be announcer.

Interval for viewing Exhibits.

Demonstration Ophthalmology, Dr. John McLean. Isotopes in Medicine, Dr. E. T. Feldsted. Arterial Insufficiency, Dr. T. R. Sarjeant.

2.00 - 4.30 p.m.

Acne, Dr. D. Williams. Obstetrical Evaluation of the Pelvis, Dr. A. E. Trites. Hæmatology, Dr. A. W. Perry. Safe Anæsthesia in General Practice, Dr. John Poole and Dr. Eric Webb. Plaster Techniques in Orthopædic Surgery, Dr. Fred Preston.

Tuesday, June 15, 1954—9.30-12.20 p.m.

Solitary Lesion of Lung, Demonstrator to be announcer. Interval for viewing Exhibits. Intra-Articular Injections, Dr. A. W. Bagnall. Treatment of Surgical Emergencies Dr. A. D. McKenzie and others.

2.00 - 4.30 p.m.

Techniques of Parenteral Fluid Administration, Dr. J. W. Whitelaw. Obesity, Dr. H. W. McIntosh. Clinical Demonstration of Joint Disabilities, Dr. A. S. McConkey. External Version, Dr. Jack Harrison. Oral Cancer, Dr. A. M. Evans and Staff.

Wednesday, June 16, 1954—9.00-12.00 noon.

Cæsarean Section and Resuscitation of the Newborn, Demonstrator to be announcer. Demonstration of Characteristic Gaits, Dr. Charles Gould. Examination of a Normal Newborn, Dr. A. F. Hardyment. Demonstration of Rehabilitation Procedures, Dr. W. J. Thompson, Dr. F. Patterson and Dr. Don Starr.

TO THE LADIES

EVERY CITY has a personality all its own, and whether you enjoy shopping, sightseeing, good food and entertainment or just relaxing, Vancouver has all this to offer all women visitors to the C.M.A. Meeting in June. Just to make sure your trip will be a happy and interesting one, without such worries as where to go and what to do, there will be a *Shopping Counsellors Service* trained to advise you and answer all your questions. This Service will be open all day, and although it is primarily for the ladies, men may also bring their shopping problems to us. Your counsellor guides will be Mrs. Donald S. Munroe, Mrs. E. Christopherson and Mrs. Leigh Hunt.

The Ladies' Committee has planned a luncheon and an afternoon of shopping which we hope will have a special appeal to our visitors. At noon busses will leave the Hotel Vancouver and within a few minutes the group will be transported to Vancouver's Chinatown, the second largest such community on the North American continent. Here, in the attractive Oriental atmposphere of Ming's Restaurant, a luncheon of authentic Chinese native dishes will be served. (If you are hesitant about using chop sticks, knives and forks will be provided.)

Following the luncheon, time has been allowed for shopping and sightseeing the Chinese community. This is an ideal spot to find your souvenirs or simply view the lovely oriental embroidery and hand-carved ivory ware and jewelry the merchants will have on display.

As a tourist attraction Chinatown is a city within a city having a population of 14,000 people. Here one will find quaint oriental shops with their curios and pagoda roofs.

Mark "a day in Chinatown" as a "must" when you visit Vancouver in June.

JANET SAUNDERS,
Women's Publicity

MEETINGS OF SPECIALIST SOCIETIES

British Columbia will provide the locale for the Annual Meetings of a number of Canadian specialist medical societies in June. All of them will meet in close relation to the Eighty-Seventh Annual Meeting of the Canadian Medical Association, to form a representative cross-section of Canadian medicine.

The following list represents present information on these important meetings:

Canadian Pædiatric Society.—President, Dr. G. R. Gayman, Vancouver; Secretary, Dr. J. C. Rathbun, 526 Waterloo St., London, Ont.; Meeting—Vancouver, June 10, 11 and 12.

Society of Obstetricians and Gynæcologists of Canada.—President, Dr. W. G. Cosbie, Toronto; Secretary, Dr. R. B. Meiklejohn, Suite 334, Toronto Western Hospital, Toronto. Ont.; Meeting—Harrison Hot Springs, June 11, 12 and 13.

Canadian Otolaryngological Society. — President, Dr. Robert Black, Winnipeg; Secretary, Dr. W. Ross Wright, 361 Regent St., Fredericton, N.B.; Meeting—Harrison Hot Springs, June 13, 14 and 15.

Canadian Anæsthetists' Society.—President, Dr. G. Cousineau, Montreal; Secretary, Dr. R. A. Gordon, 516 Medical Arts Bldg., Toronto, Ont.; Meeting—Vancouver, June 14 and 15.

Canadian Academy of Allergy.—President, Dr. C. H. A. Walton, Winnipeg; Secretary, Dr. T. H. Aaron, 502 McLeod Bldg., Edmonton, Alta.; Meeting—Vancouver, June 15.

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Canadian Rheumatism Association.—President, Dr. Louis G. Johnson, Montreal; Secretary, Dr. J. Bruce Frain, Winnipeg Clinic, Winnipeg, Man.; Meeting—Vancouver, June 14 and 15.

Canadian Association of Pathologists.—President, Dr. H. K. Fidler, Vancouver; Secretary, Dr. D. W. Penner, Winnipeg General Hospital, Winnipeg, Man.; Meeting—Vancouver, June 14 and 15.

Canadian Psychiatric Association. — President, Dr. G. R. MacLean, Ponoka, Alta.; Secretary, Dr. Charles Roberts, Department of National Health and Welfare, Ottawa, Ont.; Meeting—Vancouver, June 14.

Canadian Heart Association.—President, Dr. Harold Segall, Montreal; Secretary, Dr. John Keith, Hospital for Sick Children, Toronto, Ont.; Meeting—Vancouver, June 15.

Canadian Neurological Society.—President, Dr. H. H. Hyland, Toronto; Secretary, Dr. Allan Walters, Medical Arts Bldg., Toronto, Ont.; Meeting—Vancouver, June 19, 20 and 21.

College of General Practice of Canada.—Executive Director, Dr. W. V. Johnston, Toronto; Meeting—Vancouver, June 16.

The Canadian Association of Radiologists.—President, Dr. R. A. Macpherson, Winnipeg; Hon. Sec.-Treas., Dr. D. L. M. McRae, 1555 Summerhill Ave., Montreal 25, Que.; Meeting—Vancouver, June 15.

Federation of Medical Women of Canada.—Secretary, Dr. Jean Bellamy, 2455 - 29th Ave. S. W., Calgary, Alta.; Meeting—Vancouver, June 15.

The Canadian Medical Protective Association.—President, Dr. J. F. Argue, Ottawa; Sec.-Treas., Dr. T. L. Fisher, 180 Metcalfe St., Ottawa, Ont.; Meeting—Vancouver, June 16.

THE CHANGING STATUS OF MEDICINE*

GEO. F. SKINNER, M.D., *Saint John, N.B.*

THE PRESIDENT at this time has an opportunity to discuss any problems of importance to the profession in this Province. However, it is probably his duty to try to review the basic trends in the changing status of medicine, observed during his period on the councils and executives of the various professional organizations.

This naturally brings us to the question of National Health Insurance. One would think that all forms of prepaid plans had been sufficiently debated at this meeting; but in discussing the present status of our profession, there is a point of view always implied, but not sufficiently stressed in our dealing with the Government and the public, which has worried me as a paradox. I am going to attempt to analyze this paradox.

About ten years ago, the Canadian Medical Association drew up eighteen principles, which to my knowledge, still remain as our platform for any National Health Plan. The second principle reads as follows:

> "Inasmuch as the health of the people depends to a great extent upon environmental conditions under which they live and work; upon the security against fear and want; upon adequate nutrition; upon educational facilities; and upon opportunities for exercise and leisure, the improvement and extension of measures to satisfy these needs should precede or accompany any future organization of medical service. Failure to provide these measures will seriously jeopardize the success of any Health Insurance Plan."

To emphasize this definition of responsibility, one can say that after ten years it seems more important than ever; because as we face the future, the great unsolved problems of medicine with the possible exception of cancer, come under the term used years ago by George Crile: "The Diseases of Civilization". In these conditions, more than in other diseases, the health of humanity, physical, mental and emotional does not depend entirely on the success of the medical profession, but on all economic, political, religious and educational influences.

In this fundamental concept where do we stand today? To state our own position, let me quote from one of the great contemporary writers from outside of our profession, A. N. Whitehead, a recent professor of philosophy at Harvard, whose works rank with the classics of all time: "Of all the forces of civilization (not purely materialistic), that directly affect humanity, the success in the field of medicine has far out-stripped any other endeavour in recent years, and has been due to unrestricted criticism." Custom and tradition have been exposed to the strong searchlight of inductive reasoning and he believes that only fearless individualism could accomplish so much in so short a time.

And now the paradox. The greatest threat of control and regimentation from without, by State and other Boards composed mostly of laymen, comes not to the weak links of society, but to the profession most successful in its endeavour for the good of humanity.

This apparent contradiction makes it necessary to ask a question. Is our present position solely due to the adoption by Government of certain features of doctrinaire socialism, or is there a more basic reason inherent in our profession, or more specifically, in our recent spectacular progress? It is our very success and not our failure that has placed us in this position. Anyone who has analyzed the situation knows this to be true, but I do not believe that the implications of this fact have been sufficiently emphasized in our dealings with the Government and the Public.

*Extracts from the Presidential address at the Annual Meeting of the New Brunswick Medical Society, September 6 to 9, 1953.

Possibly the next point should be presented by a digression into history. Not to review the Hippocratic era, nor the Arabian period, when the spark of light from Greece was preserved and shone a little brighter, nor the few outstanding giants of the late mediæval and early modern history, but rather to consider the practitioner of fifty to seventy-five years ago, or, of one or two generations ago.

He was a very important person to his patients and to the local community, but let us not forget this community was small, and his tools were most inadequate. As a free independent individual, probably the best educated in the community, he helped the sick largely by his personality, sympathy and idealistic devotion to the welfare of his people. These were mostly spiritual or psychological contributions, and they must not be lost.

Within the life of doctors still practising, the whole outlook has changed. The scientific revolution has taken place in medicine as the natural sciences one by one bloomed into maturity. Out of physics came Roentgen; out of chemistry the Ehrlichs; out of biology Pasteur, Koch and Lister, and modern surgery the stepchild of bacteriology was born. One could dwell indefinitely on the romanticism of the success story, but at least two more accomplishments in science should be mentioned to understand future possibilities. Physics, chemistry and biology combined to produce the British School of Physiology and Bio-chemistry, and such famous men as Sherrington, Haldane, Starkling, Schaefer, Bancroft, Bayliss, Douglas and many others; as a result, internal medicine was ready for a new era. To illustrate just how recent was this last development, I can say that just thirty years ago one of these physiologists stated to me that there was then only one clinician in Britain using these scientific advances of physiology and biochemistry in clinical medicine; and that one man later returned to Canada.

Finally when William James emancipated psychology from philosophy and theology, another science was ready to make its direct contribution to human life and opened the way for the great European School of Psychiatry, and the next great battleground for health and happiness.

If we now turn to our original thesis, we can state that it is this phenomenal accomplishment of seventy-five years that has placed the medical profession at the crossroads. This profession has been asked to digest and utilize more fundamental knowledge in seventy-five years than in the previous twenty-five hundred years.

Out of this success, not out of failure, have come the two requirements that are making necessary changes in practice. Whereas, two generations ago the doctor was a helpful association, today there is so much to give it is absolutely essential that complete medical facilities are made available to all citizens; but scientific medicine is too costly for many patients as individuals in the present economic state. Consequently, organization to make adequate service available to all is necessary, and financial aid is necessary; but these two requirements must not be mistaken to mean controls and restrictions of the profession from outside sources.

There are those in our profession who state openly that we would not be in danger of outside control today, if the Canadian Medical Association and the Provincial Associations had been awake to their job. Such opinions can be due only to ignorance of the tremendous volume of work already done, or to a complete failure to realize the magnitude of the changes necessary to distribute scientific medicine to all citizens and still maintain the essential character of the profession. Another group are out and out defeatist. These individuals think that National Health Insurance inevitably means a loss of the doctors' independence and individuality, stating that any assurance from the Government that the personal doctor-patient relationship will not be disrupted, is all eyewash. This could be so, but I don't believe it is necessarily so if we constantly keep before ourselves and the public the two ideas outlined above, namely, that the present situation is due to the phenomenally rapid progress of scientific medicine and not to our failure; and also, that the spiritual and psychological contributions of the personal attributes of the doctor would inevitably be lost. Scientific medicine might flourish for a time, but the art of humanitarian medicine can live only in a free uninhibited atmosphere. Not only would the character of practice change but the quality of the personel would soon deteriorate.

We must allow no one to forget that the physician's contribution today depends upon a nicely balanced mathematical equation, with, on one side of the equation, long years of expensive training without remuneration, long hours of work and responsibility and the emotional stress inherent in dealing with human life. On the other side of the equation there is the psychological satisfaction in work worth while and the thrill of accomplishment, added to monetary remuneration above the average for a few years. But this equation does not balance without the addition of freedom of mind and freedom of expression,—he must in no sense be an employee. Alter either side of the equation ever so little and the delicate balance will be lost. The result could be something unrecognizable.

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Let us now consider a few earlier statements. The phenomenal scientific advancements in medicine in recent years have forced two issues, the economic problems of the patient and the reorganization of professional services. A division of labour has been absolutely necessary because no one man could master all the arts and sciences involved. On a national basis the training and qualifying of specialists has been accomplished in a very short period. This change has been so rapid that mistakes have been inevitable, but if we keep this historical approach in mind we should not be impatient amongst ourselves. In this program the status of the general practitioner has suffered and this undoubtedly has meant a loss for the people in that valuable personal interest of the family doctor. Fortunately, both the profession and the public recognize this fact; and it will be rectified. As most of you know this deficiency has been faced for some years and the results are beginning to show, thanks to a group of energetic and sincere members of the Canadian Medical Association. The essence of the attack on the problem is adequately shown by the debates on the term to be used, "General Practitioner" or possibly "Family Doctor" or possibly "Personal Doctor". In this connection I have only two things to say: First, we must give this movement our most enthusiastic support. Secondly, it is my belief, and here again I return to something referred to before, that the general practitioner or personal doctor will re-establish his importance when the profession and the public realize the importance of modern psychology and psychiatry, for in a large measure this will fall to him; and fortunately, the young men leaving University are much better prepared than we were.

And finally, independence of the profession is important not only for the individual physician and the individual patient but for the very spirit of civilization. To illustrate how much our leadership is necessary we need only call attention to the World Medical Association and all that it implies. And let us not forget that the personnel of the Canadian Medical Association has played no small part in that organization.

I suspect the fifth act of life should be in great cities; it is there, in the long death of old-age, that a man most forgets himself and his infirmities; receives the greatest consolation from the attentions of friends, and the greatest diversion from external circumstances.—Sydney Smith.

MEDICAL SOCIETIES

THE P.E.I. MEDICAL SOCIETY

A recent monthly meeting of the P.E.I. Medical Society was held in Summerside. The guest speaker was Dr. Carl C. Stoddard, Professor of Anæsthesiology, Dalhousie University. His subject, "Anæsthetic Emergencies—Cardiac Arrest and Newer Anæsthetic Drugs". The speaker was introduced by Dr. L. Prowse who spoke nostalgically of the many times together they had induced analgesia and even anæsthesia in various wardrooms during World War II. Before dinner a clinical session was held in the Summerside Hospital consisting of a question and answer period. Following an interesting talk on advantages and disadvantages of the newer anæsthetic drugs, a film was shown depicting the handling of sudden cardiac arrest. This was followed by a general discussion on the foregoing.

The subject of post-spinal headache was brought up several times. In discussion of prevention and treatment it was found that the anæsthetist was of little help because of his inexperience with this complication. In fact, one was definitely left with the impression that there had been only two post-spinal headaches on the Island in the last four years. One surgeon was heard to remark, as an afterthought, that he had noticed an increase in post-surgical headaches during this same period.

Several Polish songs were sung by Dr. Zielinski who was accompanied on the piano by his wife. They were much enjoyed.

The December monthly dinner meeting of the P.E.I. Medical Society was held as usual in the Charlottetown Hotel and was devoted to a discussion of the three new Health Grants. The three sub-committees presented their preliminary plans as to manner of employment of these grants. The meeting was well attended, the degree of interest high, the views varied and the discussion long and wearisome. Committees were "ad hocced" and "headed up", employees were "seconded" and the ablative "facto" was used in three ways, de facto, ex facto and ipso facto.

Considerable time was given to the question of just who would be eligible for rehabilitation. One member urged that no grandiose schemes should be entertained because of the smallness of the grant and added that he had one patient on whom the whole grant could be expended, and that she still wouldn't be rehabilitated.

A résumé of the Unit System of cost accounting for Laboratory Services was given by the President.

It was the final consensus that the employment of the grants needed much further study.

JOHN MALONEY

MONTREAL MEDICO-CHIRURGICAL SOCIETY

A novel and exceedingly interesting and informative discussion on the economic aspects of sickness was staged by the Montreal Medico-Chirurgical Society at Queen Mary Veterans' Hospital on January 8. Mr. James Muir, president of the Royal Bank of Canada and a governor of the Royal Victoria Hospital, as chairman of the five-man panel, emphasized that medical cost is an exceedingly wearisome problem. An answer has to be found or it may well be found for us in a way that we all want to avoid. In his long experience as a banker, he had known the financial problems of doctors. Few had grown rich and many were less well remunerated than the average citizen.

Mr. Robert E. Meagher, president of the Montreal Rotary Club, presented the case of the public. The opinion is growing in the public mind that there is now arrayed against him, as almost a solid bloc, the physician, the hospital and the sickness and accident insurance company. Whether this exists or not, the public is fast approaching the point where government intervention may have to be invoked. Even though the average Canadian does not like regimentation, the cost and the burden of being sick is slowly compelling him to look towards Ottawa or Quebec for relief.

One of the points emphasized by Mr. Meagher was that medical charges are prohibitively high, a feeling produced by comparing what a sickness insurance policy provides with the actual cost of an illness. "John Public" feels that the doctor should be a friend and an adviser to the patient, with a closer relationship than that which exists between the physician, the hospital and the insurance companies. Mr. Meagher felt that if socialized medicine is a distasteful thing and a bad thing for Canada, then prompt corrective action has to be taken by those primarily interested.

Dr. Walter Scriver, physician-in-chief, Royal Victoria Hospital, speaking for doctors, made the point that the attacks on high fees came from a public in which most people considered their own doctors' fees reasonable. The doctors' share of medical care cost is today less than 30%, and lower than 20 years ago.

Mr. A. H. Westbury, executive director, Montreal General Hospital, speaking for hospitals, emphasized that the answer to the hospital problem is not government control, but government reimbursing the hospitals for the full cost of care of medical indigents in the wards and the out-patient departments. Hospital deficits arise from a difference between revenue and cost for patients who cannot pay the full charge. The cost per patient per day for public patients has gone up to $15, whereas the Quebec Public Charities Act contributes only $5.50 and no government aid is available for the O.P.D.

Mr. E. Duncan Millican, executive director, Quebec Hospital Service Association, spoke about prepayment plans. More than 37% of Canada's population are now covered for hospital expenses. Cost rise has been partly due to increased admissions, now running about 139 per 1,000 population as against 96 in 1943. He called for accelerated public education. A. H. NEUFELD

CORRESPONDENCE

BEHAVIOUR PROBLEMS IN CHILDREN

To the Editor:

Dr. Williamson's useful article on Behaviour Problems in your October issue is marred by a piece of slack thinking, which is repeated so often in the lay press that, when it appears in a professional journal, it must be challenged.

Discussing punishment of children he writes: "The punishment should be related in time just as closely to the offense as possible and should not be administered in anger. I think that one of the most important points to remember is that anger has no place in the administration of punishment. The parent who vents his own spleen on a helpless child is doing irreparable harm to the child and is himself behaving poorly enough to warrant punishment."

George Bernard Shaw showed greater wisdom when he wrote, "If you strike a child take care that you strike it in anger, even at the risk of maiming it for life. A blow in cold blood neither can, nor should, be forgiven".

When an adult punishes a child there is a chance for each to learn, but they can only learn if the matter is faced squarely. When the adult hides behind his social rôle, which is as much canting hypocrisy as "it hurts me more than it hurts you, etc.", not only is he being dishonest but he is denying the child and himself the opportunity to recognize and face the mutual hatred that encompasses them. This hatred can only be dispelled by love, sustained despite the temporary quarrel.

Nothing destroys a child's faith in parents more surely than a sham of the sort that Dr. Williamson recommends. Indeed, from the good sense of most of his article, I suspect that he knows as well as I do that he is advising the impossible, but pays lip service to this fraud because it has become an accepted axiom. Otherwise, he would hardly emphasize speedy chastisement as soon after the event as possible, if he believed that anger could be avoided. There will, and should be, anger on both sides, and when this has been dissipated the grown-up will feel as sorry as the child and there will be no humbug.

Some people have ungovernable rages and they should never strike anyone. However, they are unlikely to learn from Dr. Williamson. For the rest of us George Bernard Shaw's advice to strike only in anger is good enough. You will be sorry and so will your child; the pair of you will learn something. Your child will learn that parents are fallible humans who can be both unkind and loving. You will learn to accept responsibility for actions, which, once the anger has left you, make you ashamed, and not to shelter behind even such admirable advisors as Dr. Williamson. In this way you will examine your actions more closely. The outcome may well be that you will punish less often and have less occasion to punish.

The danger of cold punishment is that no correcting shame goes with it to control the punisher.

Weyburn

HUMPHRY OSMOND,
Superintendent,
The Saskatchewan Hospital.

To the Editor:

I agree entirely with the comment of Dr. Osmond. It is my impression that any difference of opinion we have is overshadowed by a semantic problem. Like many other writers, I have fallen into the rather poor habit of using "punishment" and "discipline" interchangeably although I must confess that I know better.

What we are both saying can perhaps be written best in lay language about like this: "Anger has no place in discipline. When one has warned a child of the consequences of an act these consequences should be forthcoming exactly as promised. On the other hand, when the parent finally gets vexed to the point of explosion the explosion should be spontaneous and of long-remembered intensity."

I meant to imply as much in the statement: "If you are going to hit a child, hit him, don't tap him and snarl."

By no means did I mean to recommend a sham. No average parent could, even if he wanted to, be consummate actor enough to pretend that no anger existed when the time for corporal punishment arrives.

My thanks to Dr. Osmond for a well-taken point.

Walsh, Col. PAUL WILLIAMSON

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SPECIAL CORRESPONDENCE

The London Letter

(From our own correspondent)

RESTRICTIVE PRACTICES

Some surprise has been expressed at a suggestion emanating from the general medical services committee of the British Medical Association to the effect that the co-operation of the Ministry of Health should be sought in investigating the future number of medical practitioners likely to be required, and to relate these needs to the intake of students to the teaching schools. Is this an attempt, it is being asked, on the part of the medical profession to adopt restrictive practices for the benefit of the profession. Even if there were any clear-cut evidence that unemployment was likely to become rife among doctors during the next two or three decades, is it in accordance with the best traditions of a liberal profession to seek the aid of a Government department? Surely, it is being said, there is a real danger here of trespassing upon the independence of the universities and therefore upon academic freedom.

As *The Times* has pointed out in an editorial, "the question of how many doctors the country needs is, in fact, remarkably complicated. . . . If there is to be restriction in the number of medical students being accepted, it will certainly be regarded by the universities as yet another attack on academic freedom; and if there must be an inquiry, it should be undertaken by an independent committee".

GROUP PRACTICE

The Government's plans for encouraging group practice have now been announced. They consist of the allocation of an annual sum provisionally fixed at £100,000, for the purpose of providing interest-free loans to doctors who wish to develop group practices. The purpose for which these loans will be available are: (a) The erection of a new building for surgery premises and for the purchase of the necessary land where this is not already owned by members of the group; (b) the acquisition of an existing building and its conversion into surgery premises; (c) the conversion of a building already owned by a member of the group. The loan will normally be recoverable by quarterly deductions from the remuneration payable by executive councils to members of the group. No precise limit is set to the period over which the loan shall be repayable, but it will normally be between ten and twenty years.

This is a move on the part of the Ministry which will be widely appreciated. For various reasons the conception of health centres has become increasingly unpopular during the last few years, and the whole tendency has been towards the development of group practices. There are already sufficient of these established throughout the country (interesting details of two of these will be found in *The Practitioner*, 170: 619, 1953; and *The Lancet*, 1: 255, 1954) to show how effective they can be in improving the service which general practitioners can give to their patients while, at the same time, improving the conditions under which the practitioners themselves work.

RISING HOSPITAL COSTS

Figures just published for 1952-53 by the Ministry of Health show that the average cost of keeping and treating a patient in hospital has risen by 4½% in the London teaching hospitals, compared with the previous year, and now stands at £24 18s. 8d. The corresponding figure in provincial teaching hospitals was £18 13s. 9d. (8% increase), whilst in other general hospitals in England and Wales it was £15 4s. 9d. (13% increase). Typical costs for special hospitals were as follows: maternity hospitals £17 13s. 1d., isolation hospitals £17, children's hospitals £13 17s. 2d., orthopædic hospitals £11 13s.

DESIGN OF HOSPITALS AND LABORATORIES

The Nuffield Foundation announces the setting up of a division for architectural studies. This will be concerned with investigations into the functions and design of hospitals and a study of research laboratories. In the first instance, attention is to be devoted to the care of children in hospitals. The Royal Institute of British Architects has expressed its readiness to collaborate in this work. Studies in building science arising from the work of the division will be carried out in conjunction with the Building Research Station.

MORTALITY FIGURES

The provisional statistics for England and Wales for 1953, which have just been published, show a rise in the death rate, and a fall in the stillbirth rate and the infant mortality rate. Compared with 1952, the death rate has risen from 11.3 to 11.4 per 1,000 population, whilst the stillbirth rate (per 1,000 total live and still births) has fallen from 22.6 to 22.4. The corresponding figures for the infant mortality rate are 27.6 and 26.8 per 1,000 related live births. The stillbirth and infant mortality rates are the lowest ever recorded in this country. Another record has been set up by the death rate for the fourth quarter of 1953, which was 10.7 per 1,000 population, compared with 12.4 in the corresponding quarter in 1952. WILLIAM A. R. THOMSON

London, February, 1954.

OBITUARIES

LT.-COL. ERNEST RUDOLF BROWN, M.D., C.M., R.C.M.C., C.B.E., V.D., well known Montreal ear, nose and throat specialist and soldier, died on December 21, 1953. He was 87.

Retired from active practice for some years, Dr. Brown was one of the members of the original staff of the Children's Memorial Hospital and served for a time as assistant otolaryngologist at the Montreal General Hospital. During the First World War he served overseas with the Royal Canadian Medical Corps and was awarded the C.B.E. and V.D. for his war services.

A native of Quebec City, he studied at Bishop's College School and University and graduated from the latter with a B.A. degree. He attended the Trinity School of Medicine, Toronto and in 1894 graduated with M.D. and C.M. degrees.

He did postgraduate work in Edinburgh, Vienna and London and was for a period assistant resident medical officer at the Throat, Nose and Ear Hospital, Golden Square, London. In 1897 he returned to Canada and specialized in the treatment of ear, nose and throat ailments in Montreal.

He is survived by his widow.

DR. ELIAS CLOUSE, died on January 11 in Toronto, aged 98.

Born at Pleasant Hill, Norfolk County, he received his education at Simcoe High School, Woodstock College and Trinity College. He received his medical license from the Faculty of Physicians and Surgeons of the University of Glasgow and also the Royal College of Physicians and Surgeons in Edinburgh. He took further postgraduate work in London, England, and Johns Hopkins Hospital, Baltimore, where he studied under the late Sir William Osler. Dr. Clouse practised at Toronto and was one of

the founders of the Toronto Western Hospital. An organizer of the Empire Club, he was a past president and a life member. He leaves three sons and a daughter.

DR. FRANK HOUSTON COPPOCK died on December 23, 1953 at Eckville, Alta., aged 57. He was born at Winnipeg and during the First World War he was instructor in the air force. He graduated at Winnipeg in 1925, and started practice in Rosthern, Sask. in 1926. Later moving to Eckville in 1938.

Dr. Coppock was president of the College of Physicians and Surgeons of Alberta, and shortly before his death he had been re-elected as a member of the council of the Alberta Medical Association. He was a member of the Canadian Medical Association for 25 years as well as having served terms as a member of the council. Dr. Coppock is survived by his widow and two daughters.

DR. A. C. FALES, aged 84, died on November 24, 1953 at Wolfville. He attended Dalhousie University and Halifax Medical School and subsequently went to Harvard Medical School, Boston, from which he graduated in 1894. His wife predeceased him in 1941.

DR. W. F. GALLOW died on December 22, 1953 at Goderich. He was in his 83rd year. Born in Toronto, Dr. Gallow graduated from University of Toronto in 1895 and had practised in Goderich since 1899, except for 3 years with the R.C.A.M.C. in World War I.

He is survived by his widow and one son.

DR. JAMES ALBERT ROY GLANCY died on December 15, 1953 in Toronto. Dr. Glancy graduated in medicine from the University of Toronto in 1917 and practised in Toronto for 28 years. He leaves a widow and one son.

DR. ALEXANDER RONALD GORDON, aged 68 years, died on December 16, 1953. He was born in Durham, Ontario, in 1885. He graduated from the University of Western Ontario in 1911. He was a Mason and a vice-president of B.C. Lacrosse Association. He is survived by his widow, two sons and two daughters.

DR. F. W. GREEN, aged 77, died on December 24, 1953 in Cranbrook, B.C. Dr. Green was born in Victoria, and graduated from McGill University in 1898.

He had practised at Cranbrook since graduation except for his service in the R.C.A.M.C. in World War I.

He leaves a widow, a son and two daughters.

DR. CHARLES H. HAIR, died on December 28, 1953 in Toronto. He was born in Simcoe county and graduated in 1903 from Trinity Medical College. In 1914 he went to Toronto to practise as a surgeon on the staff of the Toronto General and joined the teaching staff of the University of Toronto. In 1934 he became medical officer of mines with the Accident Prevention association.

In 1937 he was elected president of the Academy of Medicine. He served in World War I with the Canadian Army Medical Corps in Toronto, later with the C.E.F. in Siberia. He is survived by his widow, two sons and a daughter.

DR. WILLIAM S. HARPER died in Toronto on January 8, 1953, aged 81. He was a medical graduate of Trinity Medical College in 1896. He practised in Queensborough, Port Perry and Madoc, before moving to Toronto. During the First World War he served with the R.C.A.M.C. He leaves a widow, a daughter and 3 sons.

DR. MICHAEL JOSEPH KENNEDY died in Kingston, Ontario on December 31, 1953. Born in Portsmouth he graduated from Queen's University in 1917 and served overseas during the First and Second World Wars. He had practiced in Surbury and Peterborough. He leaves a widow, 2 daughters and 2 sons.

DR. ALFRED JOHN LALONDE died in Cornwall, Ont. on December 13, 1953, aged 73. Dr. Lalonde was born at Osceola, Ont. and received his medical education at Queen's University, Kingston, graduating in 1904.

He first practised in New York City and came to Cornwall in 1910. He was a member of the Cornwall Medical Association and of the medical staff of the Hotel Dieu and Cornwall General Hospital and since 1946 was physician and surgeon for the Department of Indian Affairs (St. Regis Reservation). He leaves a widow and 2 sons.

DR. DONALD C. McFARLANE, died in London, Ont. on January 4, 1953, aged 60. He was born in Dorchester, graduating in 1916 from the medical school at UWO here and entering the Royal Canadian Army Medical Corps, with which he served overseas with the rank of captain until 1919. After postgraduate study he returned to London in 1925 and was appointed to the staff of the medical faculty of UWO. In 1946 he became professor and head of the medical school's department of ophthalmology, otology, rhinology, and laryngology. Dr. McFarlane was also chief of eye, ear, nose and throat services at Victoria Hospital.

He was elected a Fellow of the Royal College of Surgeons (Canada) and a Fellow of the American College of Surgeons.

He was a member of the council of the Canadian Otolaryngological Society and the council of the Canadian Ophthalmological Society, and the American Academy of Ophthalmology and Otolaryngology. He is survived by his widow and 2 sons.

DR. ARCHIBALD MacINNIS, aged 83, died at Toronto General Hospital in December, 1953. He was born in Grey County, Ont. and graduated from the University of Toronto.

DR. HORATIO CLARENCE NORQUAY of Selkirk died on December 27, aged 84. Born at High Bluff, he graduated in 1895 from Manitoba Medical College, and practised at Selkirk, with the department of Indian affairs at Norway House and at Battleford, Sask. In 1938 he retired and has lived in Selkirk. He is survived by his widow, three sons and two daughters.

DR. J. ALBERT PREFONTAINE, died in Sherbrooke on December 18, 1953 at the age of 46. Born at St. Marguerite Station, he studied at Laval University, Quebec, where he graduated in 1933. He practised in Coaticook and Sherbrooke but also went to the Medical School in New York. He is survived by his widow.

DR. LLOYD STEINER, died at Tulare, California, on January 4, 1954, from a motor accident. He was 29. Born and educated in Toronto, Dr. Steiner graduated in medicine from the University of Toronto in 1953 and interned at the Royal Columbian Hospital, New Westminster. He was a Flight Lieutenant with the RCAF in 1944 and 1945. He is survived by his widow.

DR. JOHN LESSLIE UREN, aged 52, died on December 24, 1953. He was on the board of governors of Physicians Services Inc., on the council of the Academy of Medicine and formerly on the staff of Toronto General Hospital. Born in Toronto, Dr. Uren attended Normal Model School, Jarvis Collegiate and University of Toronto Schools, where he was gold medalist. In World War II he served in the R.C.A.M.C. with the rank of major. Surviving are his widow, a daughter and a son.

DR. JENNIE GRAY WILDMAN, died in Barrie on December 16, 1953 at the age of 90. She was born in Dundas. Well known as a former member of the consultant staff of the Women's College Hospital, Toronto, she moved to Barrie in 1928. Dr. Wildman was one of the first graduates of Women's Medical College, Toronto,

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and received her degree of M.D.C.M. at Trinity University in 1892. Dr. Wildman was head of the original dispensary where, in 1911, the history of Women's College Hospital began.

DR. F. D. WILSON, aged 69, died on December 20 in Calgary. He was born in Simcoe County, Ont., and graduated from the University of Toronto in 1908 after which he served with the Toronto General Hospital for two years. Forty-three years ago, in 1910, Dr. Wilson moved to Calgary and for a time was chief surgeon at the Calgary General Hospital before entering private practice.

Dr. Wilson found time for active participation in the Canadian Medical Association, the Alberta College of Physicians and Surgeons, the Calgary Medical Association, the Ranchmen's Club and the Golf and Country Club. His wife predeceased him in 1944. Surviving is a daughter.

DR. FRANK LESLIE WOODMAN, died in Edmonton on January 1, He was 35. Dr. Woodman was born in Calgary. He was educated at the University of Alberta, and had practised medicine at Westlock since 1945. He is survived by his widow, a daughter and a son.

ABSTRACTS *from current literature*

MEDICINE

Some Basic Considerations of Obesity as a Public Health Problem.

JOLIFFE, N.: AM. J. PUB. HEALTH, 43: 989, 1953.

Many persons are living longer, eating too much, and getting too fat. The end result will be death from arteriosclerosis and other degenerative diseases. Labour saving devices were few and far between in 1900, everyday living called for the expenditure of greater physical energy than now. At that time the per capita consumption of calories in food was 3,100 a day. This remains about the same today. 2,870 calories per capita a day is a comparable maximum for an older and less hard-working population.

It has been estimated that 25 to 30% of the population is overweight, and since it is true that death due to arteriosclerosis and other degenerative diseases occurs earlier in the obese than in the non-obese the problem is clear. If you are overweight, reduce, and—more important—stay reduced.

Basic information regarding obesity follows: (1) "Obesity is invariably caused by a greater intake of calories in food than expenditure in energy." (2) "The essential component of every reducing regimen is a caloric deficit. A deficit of 3,500 calories leads to the loss of a pound of adipose tissue." Exercise is exaggerated as a means of losing weight. For instance, in order to lose 2 pounds one would have to walk 10 miles a day for a week without increasing his food intake. This is impossible because the appetite would be greatly stimulated. The obese person needs to eat 1,000 calories a day less for a week in order to lose 10 pounds. This can be done without additional exercise. (3) "The reducing diet should form the basis of dietary re-education, so that proper eating habits will continue after the desired loss of weight has been attained." Trick diets and prescribed menus aren't helpful. The obese person should learn to assign caloric quotas to standard portions of all foods. They should know food values and just what constitutes the balanced meal. Thus, they won't be caught short when they are away from home and their menu for the day. A little practice, and calory counting will become second nature.

ISABEL M. LAUDER

Observations on the Use of Cortisone and ACTH in Rheumatoid Arthritis.

CLARK, W. S., TONNING, H. O. KULKA, J. P. AND BAUER, W.: NEW ENGLAND J. MED., 249: 635, 1953.

A careful study is presented of 52 patients with rheumatoid arthritis treated with cortisone or ACTH in addition to other routine methods of clinical management of the disease. Cortisone induced major subjective improvement in 74% of subjects to whom it was administered, with major objective improvement being evident in 38%. With ACTH major subjective improvement occurred in 60% with major objective improvement in 22%.

The authors conclude that cortisone and ACTH can favourably alter the clinical and laboratory manifestations of rheumatoid arthritis. Subjective improvement is greater than objective results and histologic evidence of joint inflammation persists even after prolonged therapy. All patients do not respond favourably and in some progression of the disease will occur despite adequate hormone administration.

Hormone therapy should not be employed in rheumatoid arthritis until maximum benefit from conservative measures has been established and should always be combined with such measures. Improvement which results with cortisone or ACTH is frequently the direct effect of rehabilitation procedures facilitated by the period of hormone administration. There is as yet no evidence that either agent alters the remission rate or the natural course of the disease. NORMAN S. SKINNER

Detection of Achlorhydria Without Intubation.

CONWAY, H. AND MEIKLE, R. W.: BRIT. M. J., 2: 1019, 1953.

Intubation of the stomach to obtain samples of gastric juice for examination has two major disadvantages: it often proves distressing to the patient, and the act of swallowing the tube tends to alter the composition of the juice. It has been shown in dogs that nausea and retching inhibit the secretion of gastric hydrochloric acid even when histamine is injected every ten minutes, and the presence in the stomach of excessive saliva reduces the acid content.

The clinician wants to know from a gastric analysis whether the patient's stomach is able to secrete hydrochloric acid. This information can now be obtained by a simple test which does not involve the passage of a tube. Patients must not ingest vitamins or drugs containing calcium, magnesium, iron, or aluminium during the previous 24 hours. After a fast of 8 hours, the bladder is emptied and the urine is saved. Immediately afterwards, 0.5 mgm. of histamine acid phosphate is injected subcutaneously and 2 grams of diagnex (a cation exchange indicator compound), in four ounces of water, are given orally with instructions to avoid chewing the granules. At the end of one or two hours, the bladder is emptied and the volume of all three urine specimens is measured; each is treated with ether and sulphuric acid for the extraction of quinine. The final acid extracts are compared with a standard solution of quinine sulphate in a Hilger fluorimeter. The natural fluoresence of the night urine expressed as μg. of quinine, and the amount of quinine in each hourly specimen are then calculated.

In this series, the results in 22 patients agreed well with those obtained by the aspiration technique, but a discrepancy occurred in a patient who had a partial gastrectomy.

At present, the diagnex test is likely to be used only in selected patients, especially in those who object to swallowing a stomach tube. Eventually, however, it may become the routine method of gastric analysis.

B. L. FRANK

The Effect of Penicillin Prophylaxis on Streptococcal Disease Rates and the Carrier State.

WANNAMAKER, L. W., DENNY, F. W., PERRY, W. D. RAMMELKAMP, C. H., ECKHARDT, G. C., HOUSER, H. B. AND HAHN, E. O.: NEW ENGLAND J. MED., 249: 1, 1953.

A carefully controlled study of the effect of penicillin on Group A streptococci carriers among army personnel showed eradication of the organism with oral dosage of 1,000,000 units twice daily for ten days. Suggestive evidence was obtained that probably half this dosage would be effective but trial with 250,000 units once daily for ten days did not suppress or eradicate the carrier state in many subjects. Eradication of the organism was not effected by a single intramuscular injection of 600,000 units but four such injections on alternate days was practically uniformly effective. Benzethacil was effective in a single injection of 600,000 units.

The oral administration of penicillin in dosage of 1,000,000 units twice daily for five to ten days effectively reduced carrier and disease rates in large groups of men and provides a reasonably practical method for the control of epidemic streptococcal disease. No serious complications resulted from the use of the penicillin, diarrhœa was common, moderate laryngeal œdema occurred in one subject and skin reactions were noted in approximately 1%. NORMAN S. SKINNER

The Prognosis in Gastric Ulcer Treated Conservatively.

BANKS, B. M. AND ZETZEL, L.: NEW ENGLAND J. MED., 248: 1008, 1953.

The differentiation between benign and malignant gastric ulcer is a very difficult clinical problem and a very important one since about 10% of gastric ulcers ultimately turn out to be malignant. In order to determine the safety of good medical management the records of the Beth Israel Hospital, Boston, were reviewed over the period 1931-1946 and 48 cases selected for study which fulfilled all the criteria of having been adequately treated and adequately followed. Eight of these patients died of carcinoma of the stomach even though all had been considered to be benign ulcer.

The proper management of gastric ulcer consists of immediate admission to hospital for evaluation and decision regarding therapy. Conservative treatment should be continued only if there is progressive diminution in size of the gastric ulcer on repeated radiologic examination and the patient should not be discharged from hospital until the lesion has completely disappeared and the gastric wall is normally pliable in the previously ulcerated area. Subsequent medical management must include a regular semiannual radiologic examination of the stomach for three years and then annually thereafter. If this rigid program is carried out the risk of death from carcinoma of the stomach equals the mortality rate of gastric resection. If such a program is unlikely to be adequately followed operation would probably be indicated. NORMAN S. SKINNER

SURGERY

Experiences with Cardiopericardiopexy in the Treatment of Coronary Disease.

THOMPSON, S. A. AND PLACHTA, A.: J. A. M. A., 152: 678, 1953.

For the past 13 years, the senior author has been treating a selected group of coronary patients with this surgical procedure. The operation consists in spreading sterile powdered magnesium silicate over the myocardium inside the pericardial sac. This substance is known as U.S.P. talc; however, it is not the ordinary commercial variety of talcum powder, but is a form of powdered silica. It is believed that this procedure converts the ischæmic myocardium of coronary disease into a hyperæmic myocardium. When the talc is placed inside the pericardial sac, it acts as an irritant and causes three principal reactions; a talc granuloma is produced that involves the superficial surface of the myocardium; it stimulates the development of interarterial coronary anastomoses; and it produces adhesive pericarditis. This operation has been carried out by these workers on 57 patients, with 7 deaths (12%). Of the 50 patients who survived, 5 (10%) have received less than 50% improvement and are classed as poor results. The remaining 45 patients (90%) have received more than 50% improvement, and some of these have experienced more than 75% improvement. Improvement is gauged by four criteria: First, decrease in the amount of anginal pain; second, the increase in the exercise tolerance; third, the improved ability to attend to daily needs; and fourth, a return to the former or some other gainful occupation. It was emphasized that most of these patients were medical failures and that many were completely incapacitated and some in a terminal state, prior to operation. The writers feel that this treatment is of distinct value in coronary disease. S. J. SHANE

Use of ACTH and Cortisone in Surgery.

COLE, W. H., GROVE, W. J. AND MONTGOMERY, M. M.: ANN. SURG., 137: 718, 1953.

The temporary improvement in a patient's condition brought about by the use of ACTH and/or cortisone may make a surgical operation possible that could not be attempted otherwise. In usual dosage it is doubtful that these drugs have any detrimental effect on wound healing. The increase in appetite and euphoric state, frequent decrease in pain as well as the adverse effects of long continued use are discussed.

Malnutrition as a cause of inoperability may be improved by ACTH and cortisone and cases of carcinoma of the rectum and of the lung which improved so greatly in 5 days that successful resections were possible are illustrative. In certain postoperative conditions, particularly hypotension, complications can be dramatically improved by intravenous ACTH, but of course it should not be used for shock due to blood loss. Thrombocytopenic purpura is temporarily improved by ACTH, and so are certain other purpuras, and a response to the drug may indicate a response to splenectomy. A high proportion of cases of ulcerative colitis are temporarily improved by ACTH which seems more effective than cortisone. It is suggested that the increased appetite induced is the only benefit these drugs have on burned patients unless they have true adrenal cortical insufficiency. The prevention of anaphylactic shock such as from the rupture of an echinococcus cyst during its removal may be useful.

Other uses are discussed: in regional ileitis, acute pancreatitis, non-suppurative tenosynovitis, Dupuytren's contracture, abdominal adhesions, thyroid crises, pulmonary œdema and thrombophlebitis. Contraindications to the use of ACTH and cortisone include diabetes mellitus, peptic ulcer, tuberculosis, Cushing's syndrome, acne vulgaris, hypertensive cardiovascular disease, chronic nephritis and pregnancy. BURNS PLEWES

Carcinoma of the Colon and Rectum.

ASCHERMAN, S. W.: ARCH. SURG., 66: 208, 1953.

A review of 461 post mortems at Cook County Hospital was undertaken in an effort to supplement the knowledge of carcinoma of the large bowel. Over 70% of the cases of rectal carcinoma were male, and of carcinoma of the colon 60% were male. There is no difference in tendency to metastasize in different sites. The presence of tumour cells within a vein is not proof that distant metastases have occurred. Retrograde lymphatic metastases may occur by the blocking of normal lymph flow. Large bulky tumours tending to grow into the bowel lumen are usually slow growing. Such tumours are more common in the right colon and show a better five-year survival rate. Obstructive lesions have a lower survival

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rate and metastases are more extensive. Colicky pain and rectal bleeding were the most common reasons for seeking hospital aid. Weight loss was hard to evaluate but was very common. Change in bowel habit occurred in 60%. Of all cæcal carcinomas 45% were palpable, but only 5% of all colon carcinomas were palpable by abdomen. Iron-deficiency anæmia was evident in 50% of right colon lesions. Hæmorrhoids were associated with 36 cases of carcinomas of rectum or sigmoid. Ulcerative colitis was associated with multiple carcinoma in 5 cases and sidetracking operations did not seem to lessen the incidence. Local recurrence after operation was higher in the rectum and recto-sigmoid. BURNS PLEWES

Anomalous Peritoneal Encapsulation of the Small Intestine.

THORLAKSON, P. H. T., MONIE, I. W. AND THORLAKSON, T. K.: BRIT. J. SURG., 40: 490, 1953.

Three cases are described in which, at laparotomy, the small intestine lay behind a translucent membrane. In one case the greater omentum was fused with the peritoneum of the edge of the liver. In another, the membrane was thick and adherent to the anterior abdominal wall and part of the jejunum had undergone partial volvulus causing obstruction. In each, the operation which disclosed the condition was done for an unrelated condition so that the anomaly *per se* produced no symptoms or signs.

The abnormal development of the midgut is discussed and illustrated by diagrams. Only two references to such a congenital anomaly were found in the literature.

BURNS PLEWES

The Relation of Gastric Ulcer to Carcinoma of the Stomach.

MARSHALL, S. F.: ANN. SURG., 137: 891, 1953.

In a group of 411 cases of gastric ulcer, the incidence of malignancy was 15.8%. There is insufficient evidence that gastric ulcers become malignant often, but ulcerocancer may be frequently mistaken for benign ulcer. In a considerable number of patients dangerous delay results when a gastric ulcer is thought to be benign. Complete healing by medical treatment in hospital is a necessary therapeutic test. Malignant ulcers may improve greatly both clinically and radiologically, but incomplete healing or recurrences must be resected promptly. Criteria as to the size of the ulcer, location of the lesion and age of the patient are not completely reliable. Routine surgical treatment of all cases of uncomplicated gastric ulcers is not recommended, but probably 50% or more of gastric ulcers should be resected since 15.8% have been found to be malignant. Temporizing with non-healing ulceration lesions of the stomach involves serious risk to the patient and is completely unjustified.

In discussion, Dr. Frank Lahey spoke of the difficulty in persuading doctors that gastric ulcers are likely to be malignant. Every patient with gastric ulcer must be x-rayed again in a month, then in 2 months and if this is negative let him go 3 months, then if negative, 6 months. Every case that reopens or fails to close should be operated upon. Every patient with a gastric ulcer should be told that he has a 15% chance of having cancer that may become inoperable if he goes on with it.

Dr. Randin reiterated, both in his paper and in discussion that the patient with gastric ulcer should be sent to the surgeon, that it is the only way we can improve the end results of gastric cancer. He emphasized that serial sections must be cut through these ulcers to find early cancers. He stated that he was operating upon every one of the gastric ulcers sent to him.

BURNS PLEWES

An Improvement of 180% in the Five-Year Survival Rate of Patients with Carcinoma of the Stomach.

WALTERS, WALTMAN AND BERKSON, J.: ANN. SURG., 137: 884, 1953.

At the Mayo Clinic the five-year survival rate after the diagnosis of carcinoma of the stomach between 1908 and 1916 was 5%, and between 1940 and 1949 it is 14%. The improvement is due to an increased laparotomy rate, increased resectability rate and decreased operative mortality rate. Total gastrectomy is being done much more often in recent years with encouraging results. Improvement in survival rates for those who survived resection are also shown: from 29.2% for 5 years and 21.7% for 10 years in the 1907-1916 series to 34.8% and 26.7% respectively in the 1940-1949 series. These figures do not show the increased extension of benefits of surgical intervention during the past 9 or 10 years to a larger proportion of elderly patients who have carcinoma of the stomach. Many more patients between 70 and 79 years have undergone gastric resections in recent years with no greater mortality than patients a decade younger. This has been possible by better and longer preoperative preparation, better anæsthesia, better postoperative care and a change in the attitude of the surgeon who starts every operation with the intent of doing a wide resection.

Five year survival rates following operation of 48.5% if no lymph node metastasis are present and of 18.6% if lymph nodes are involved, may be expected. After five years the survival rates practically parallel the normal population.

Further improvements are possible. Gastric polyps should be removed. About 10 to 20% of gastric ulcers are malignant, so that the physician assumes a great responsibility who treats a gastric ulcer medically.

BURNS PLEWES

Cysts of the Semilunar Cartilages of the Knee-Joint.

BONNIN, J. G.: BRIT. J. SURG., 40: 558, 1953.

Cysts of the knee cartilage are like ganglions elsewhere but recur more frequently after removal. They are a proliferative lesion rather than a degenerative one. Trauma is precipitating in one-third, a minor part of the history in another third and absent in the remainder. A cyst may contribute to the formation of a tear. A triad of clinical features is noted in cysts of the menisci: pain after exercise relieved by rest but often worse at night, cystic swelling over the joint line, localization of pain and tenderness on pressure on the joint line. A small proportion of bilateral cases involve the lateral meniscus. Unilateral cases involve the medial cartilage more frequently. Effectual cure follows complete removal of the cartilage. Twenty-nine cases are described.

BURNS PLEWES

GYNÆCOLOGY AND OBSTETRICS

The Place of Forceps in Present-Day Obstetrics.

JEFFCOATE, T. N. A.: BRIT. M. J., 2: 951, 1953.

There is danger in praising forceps too highly and in claiming too large a place for them; it might encourage their abuse. There is also danger in adopting an ostrich-like attitude and failing to recognize that times have changed, and are changing. It will not do to condemn forceps merely on the grounds that they represent interference with Nature, if it can be shown that their results improve on those achieved by Nature.

The fact that childbirth is a physiological process does not necessarily mean that it is a benign one. The reproductive function in all species is both dangerous and wasteful. Nature is not concerned with making reproduction safe for individual mothers or offspring; she expects losses and for this reason provides a supply of potential

offspring which is so bounteous as to offset them and ensure the continuance of the race.

The natural maternal and fetal mortality rates are unknown, but it is quite certain that they are much higher than those which obtain when the medical and nursing professions stand by ready to interfere judiciously with Nature. Obstetricians do well to leave childbirth to Nature only so long as she is efficient, but when she fails—as she often does—lives can be saved only by carefully planned and skilful intervention. Moreover, as the means and methods for intervention become safer, they can offer more and more to the individual than can Nature. ROSS MITCHELL

The Fetal Risk in Breech Presentation.

DALEY, D. AND MICHAEL, A. W.: J. OBST. AND GYNÆC. BRIT. EMP., 60: 492, 1953.

The generally reported breech mortality figures are unnecessarily high and with careful assessment of each case delivery in hospital conducted by a registrar or consultant will usually result in spontaneous delivery of a live child in the uncomplicated cases. In such circumstances, the mortality should not be appreciably higher than for vertex delivery and may well be lower than if the mother is subjected to version under anæsthesia. Elective Cæsarean section can be reserved for those with extremely large infants or some associated complication, but the wider use of section in labour for fetal distress associated with early prolapse of the cord and the more severe degrees of uterine inertia should lower the mortality still further.

A plea is made for an exact definition of what constitutes a complication of breech delivery, so that hospital reports and reviews of large series will be comparable at a glance. ROSS MITCHELL

The Relationship of Hormonal Environment to the Genesis and to the Inhibition of Neoplastic Growth. Is Cancer Autonomous?

RAWSON, R. W.: AM. J. OBST. AND GYNEC., 66: 999, 1953.

It has been demonstrated in various laboratories that the administration of œstrogenic hormones to experimental animals results in the production of tumours of the following tissues: breast, cervix, uterus, testes and lymph nodes. It has also been demonstrated that the administration of pituitary hormones or the induction of increased elaboration of certain pituitary hormones results in the development of uterine placentomas, ovarian, lymphatic, lung, adrenal, breast, testicular, and thyroid tumours.

It has been amply demonstrated that by altering the hormonal environment the following human tumours can be made to recede or to function like normal tissues: prostate, breast (male and female), lymphatic, and thyroid. These observations are being further investigated for clues which may ultimately lead to physiologic methods of controlling the development and course of such tumours. ROSS MITCHELL

The Treatment of Female Genital Tuberculosis with Streptomycin and Para-aminosalicylic Acid.

SERED, H., FALLS, F. H. AND ZUMMO, B. P.: AM. J. OBST. AND GYNEC., 66: 823, 1953.

Eighteen consecutive cases of genital tuberculosis treated with PAS and dihydrostreptomycin are presented. The average dose of streptomycin was 1 gm. per day given in a single intramuscular injection. The dosage of PAS was 10 gm. per day. The duration of combined drug therapy was as a rule 3½ to 4 months.

The side reactions and toxic behaviour of PAS are discussed, and the management of these complications is considered in detail. PAS may be used for its suppressive effects long after the tubercle bacillus has become resistant to streptomycin. The use of PAS has lessened the possibility of relapses and recurrences.

The clinico-pathological peculiarities of tuberculosis permit diagnosis of that disease to be easily missed. Abdominal distension, with or without ascites, appearing within a few months after abortion or delivery strongly suggests the possibility of genital tuberculosis.

The advantages of combined anti-microbial therapy are outlined. The recent introduction of isonicotinic acid hydrazines does not minimize the value of combined PAS and streptomycin therapy. ROSS MITCHELL

Adrenal Hæmorrhage in Pregnancy.

PETERSON, W. F. AND GOLDZIEHER, J.: AM. J. OBST. AND GYNEC., 66: 648, 1953.

Adrenal apoplexy in the adult has been considered a rare finding, only 34 cases being reported in pregnancy. Another case is reported. Adrenal physiology in pregnancy is discussed. Apoplexy may occur when stress is imposed upon the adrenal cortex already carrying the burden of pregnancy. Adrenal hæmorrhage with acute insufficiency may occur during complicated pregnancies more frequently than is realized. The clinical picture of adrenal hæmorrhage is outlined and a plan of therapy advanced. ROSS MITCHELL

Poliomyelitis and Pregnancy.

COBB, S. W., STUART, J. AND MENGERT, W. F.: J. OBST. AND GYNEC., 2: 379, 1953.

The incidence of poliomyelitis among pregnant women is apparently increasing. Congenital malformation does not seem to occur with poliomyelitis as with rubella. There is not much evidence that the virus of poliomyelitis can cross the placental barrier.

The medical management of the pregnant woman with acute or chronic poliomyelitis is identical with that of the non-pregnant. In general, the rule should be: Ignore the pregnancy, treat the disease.

The labouring patient must be delivered as expeditiously as possible, reserving Cæsarean section for obstetric indications. ROSS MITCHELL

PÆDIATRICS

Sudden and Unexpected Death in Infancy.

JUDGE, D. J.: POST-GRAD. MED., 14: 79, 1953.

One of the greatest tragedies for both the physicians and parents is the sudden and unexpected death of an infant. The parents, fearful that possible errors on their part have led to the death, turn to the physician for reassurance and explanation.

The main causes of such infant deaths are listed as follows: (1) Aspiration of fluids. Vomitus, formula, excessive secretions, foreign bodies or amniotic fluid may be aspirated and will account for a large number of these deaths. The advisability of placing the infant in the face-down position to allow for dependent drainage is important. This position has been exaggerated as the cause of mechanical suffocation. (2) Infection is the next most common cause. Pneumonia proceeds so rapidly in the very young, as in the old, as to present few, if any symptoms to the unpractised eye. Aspiration pneumonia can be placed in this category. General sepsis is also very often missed and can become a fulminating matter in a few short hours. (3) Intracranial hæmorrhage following a difficult passage through the birth canal, or a Cæsarean can lead to sudden death. It is so unexpected because so often the infant seems to be in good condition after delivery. Vigorous resuscitative attempts can lead to intra-abdominal hæmorrhage or sudden circulatory collapse. (4) Suffocation is not as great a hazard as it once was thought to be. Faulty sleeping equipment can be the cause here. One author reports that of 318 cases thought to be due to this, after autopsy only 25 were found to have really suffocated. (5) Thymic disorders. The thymic gland has been greatly overrated as a cause in these deaths. (6) Congenital anomalies of the

heart, great blood vessels, lungs, digestive tract and brain is often a cause of these unexpected deaths. (7) Sensitivities to the anæsthetic or preoperative drugs may lead to sudden death.

Of great importance, concludes the author, is the detailed and complete autopsy of every such infant death. ISABEL M. LAUDER

Prophylaxis of Allergic Disease in the Newborn.

GLASSER, J. AND JOHNSTONE, D. E.: J. A. M. A., 153: 620, 1953.

Many babies, especially those with an immediate family history of allergy, exhibit food sensitivities more frequently in their first months than at any other period in the life span. This is attributed to the absorption of unaltered proteins from their intestinal tracts. These newborns are still physiologically and immunologically immature.

The authors have previously proved the practicality of feeding infants from birth on a diet in which soy-bean milk completely replaced cow's milk. In this research, they have attempted to show that if cow's milk is withheld immediately from these potentially allergic infants, and the soy-bean milk formula substituted (unless breast-fed) the development and magnitude of major (asthma, etc.) and minor (atopic dermatitis, etc.) allergies will be greatly lessened. This influence will be felt at a later age too, even after cow's milk has been added to the diet.

The experimental group consisted of 96 infants with allergy in the immediate family. The mothers' diets were restricted during the prenatal period, and such foods as cheese and eggs were eliminated. This restriction was continued postnatally for the nursing mothers. Calcium and phosphorus was furnished by medication. One pint of milk, boiled 10 minutes, per day was allowed. The babies were immediately placed on a soy-bean milk (1/3) and water (2/3) formula. This was increased to equal parts after the infant had become accustomed to it. Cow's milk was introduced into the diets at varying periods from 1 to 9 months, with 50 of the babies receiving it from 6 to 9 months. The allergic histories of the subjects were received periodically, *i.e.*, 7 to 12 months (15), 1 to 2 years (14), 2 to 5 years (45), and 5 to 10 years (22).

Two control groups were used in comparing results. One consisted of 65 siblings of infants in the experimental group, and the other of 175 subjects chosen from 1,215 allergic families and carefully screened so that background of experimental infants and controls closely resembled one another.

As far as they have been followed, comparisons show that the infants in both of the control groups had four times as many allergic diseases as in the research group. This promises the possibility of applying the general principles of prophylaxis to the immunologic and allergic management of potentially allergic children. However, as the authors point out, if the infant is given cow's milk once after birth the harm is done.

ISABEL M. LAUDER

Rôle of the Pædiatrician in Prevention of Needless Neonatal Deaths.

CLIFFORD, S. H.: J. A. M. A., 153: 466, 1953.

Although premature infants are more vulnerable to the causes which lead to death of full term infants, prematurity *per se* is no longer acceptable as a cause of death. These infants while representing only 7 to 8% of all births, contribute 2/3 of total neonatal mortality. The pædiatrician has the responsibility of applying all his knowledge to reducing this figure; of protecting his charges so that they may survive this hazardous periods without resulting handicaps. He must also disseminate his knowledge so that babies everywhere may benefit.

The author points out that even though the most critical time for the infant is in the delivery room, the pædiatrician does not often see his charges until several hours after birth. He points out that very "sleepy" babies resulted in the pre-anæsthesia-analgesia days—the time of home deliveries. Now, 5 to 10% "sleepy" babies are so slow in breathing that resuscitation is deemed advisable. The pædiatrician is alarmed lest too heroic methods are undertaken and the infant may suffer more than if left strictly alone. For instance, bronchoscopy should not be attempted, it is seldom necessary since those with a large experience in performing autopsies on the newborn have not encountered laryngeal or bronchial plugging which could not be relieved by direct suction.

All infants should be watched closely for symptoms of respiratory distress. These are prone to develop (1) after Cæsarean; (2) difficult delivery; (3) intrauterine anoxia; (4) infants delivered of diabetic mothers; (5) all prematures. Air passage should be cleared by postural drainage. The stomach must be aspirated of its fluid content, and the infant placed in an incubator with 40 to 50% oxygen. Since infections are likely to occur they should be put on antibiotic therapy, either alone or in conjunction with chemotherapy. Since these babies are comatose oral feeding or fluids are not indicated as it might prove disastrous through further aspiration.

Symptoms pointing to injury at birth, congenital malformations, infections (epidemic diarrhœa) and blood dyscrasias should be carefully watched for and prevented when possible. ISABEL M. LAUDER

THERAPEUTICS

Effects of Hexamethonium Bromide on the Cerebral Circulation in Hypertension.

DEWAR, H. A., OWEN, S. G. AND JENKINS, A. R.: 2: 1017, 1953.

The fall in total peripheral resistance which occurs after the administration of the ganglion-blocking agent hexamethonium bromide is not distributed equally between the vascular areas of the body; there follows, therefore, a redistribution of peripheral blood flow. For instance, the flow to the foot and toes is greatly increased, while that to the calf segment of the leg muscles is diminished; vascular resistance in the liver decreases enough to maintain the blood flow at its previous level.

The effects upon the cerebral and coronary circulations are of particular importance. It was shown experimentally that the mean coronary blood flow is maintained. The cerebral blood flow was estimated by a modification of the nitrous oxide method in six patients suffering from arterial hypertension before and after administration of hexamethonium bromide. The mean arterial blood pressure fell by an average of 30%, but no significant change occurred in the mean cerebral blood flow. In four of these patients the rate of cerebral oxygen utilization was measured and showed no essential alteration. It seems unlikely that hexamethonium lowers the abnormal cerebral vascular tone in hypertension by blocking sympathetic impulses to the cerebral arteries.

The findings of this work imply that cerebral nutrition and metabolism will not ordinarily be impaired by the treatment of hypertensive patients with hexamethonium bromide. The possibility of an increased likelihood of cerebral thrombosis in patients in whom arterial disease favours the occurrence of such a complication can not be considered to be eliminated. B. L. FRANK

Isoniazid in Combination with Streptomycin or with P.A.S. in the Treatment of Pulmonary Tuberculosis.

Fifth Report to the Medical Research Council by their Tuberculosis Chemotherapy Trials Committee: BRIT. M. J., 2: 1005, 1953.

As part of the clinical trial carried on with isonicotinic acid hydrazide in the treatment of pulmonary tuberculosis, 391 patients were studied in 50 hospitals. There were three main groups of patients: Group 1, acute rapidly progressive disease of recent origin; Group 2,

other forms of pulmonary tuberculosis suitable for chemotherapy; Group 3, chronic cases who were considered unlikely to respond to chemotherapy.

After three months' treatment a comparison was made between the patients in Group 1 and Group 2 on streptomycin, 1 gram daily, plus isoniazid, 200 mgm. daily, and those on sodium P.A.S., 20 grams daily, plus isoniazid, 200 mgm. daily. There was a similar distribution of patients with severe and less severe disease at the beginning of the treatment in these two groups.

At the end of three months, 89% of the patients treated with streptomycin plus isoniazid showed improvement of their general condition and 88% of the patients treated with sodium P.A.S. plus isoniazid. The average gain in weight during this period was 12.8 pounds in the former and 10.8 pounds in the latter series. The temperature fell to normal in 79% of febrile patients in the first group and 85% of febrile patients in the second group. Patients with an E.S.R. of 21 or more before treatment showed a reduction of the rate to 10 or less in 37% of the first series and in 46% of the second series. A radiologist unaware of the treatment which patients received reported on radiographic appearances: 54% of the first series and 42% of the second series showed two-plus or three-plus improvement; there were two radiographic deteriorations and there was one death with each treatment. Bacteriological examination after 3 months showed that 65% of the first group and 66% of the second group were negative on direct examination and on culture. Bacillary resistance to isoniazid was found in two out of 39 culture-positive patients of the first group compared with none of 29 similar patients of the second group; bacillary resistance to streptomycin was found in one of 38 culture-positive patients of the first group and in one of 28 similar patients of the second group.

Judging from the results after three months, it appeared that sodium PAS, 20 grams daily, plus isoniazid, 200 mgm. daily, was a very effective combination, both clinically and bacteriologically; it was thought to rank with the most efficient methods of treatment so far investigated, namely streptomycin, 1 gram daily, plus isoniazid, 200 mgm. daily, and streptomycin, 1 gram daily, plus sodium PAS, 20 grams daily. Patients on streptomycin plus isoniazid showed a little better gain in weight, and a higher proportion showed substantial radiographic improvement.

Regarding sensitivity, it was concluded that sodium PAS, 10 grams daily, plus isoniazid, 200 mgm. daily, may prove to be a bacteriologically effective combination for at least three months. Streptomycin, 1 gram twice a week, plus isoniazid, 200 mgm. daily, appeared to be less effective than streptomycin, 1 gram daily, plus isoniazid, 200 mgm. daily, in preventing the development of bacterial resistance to isoniazid over a 3-month period.

B. L. FRANK

INDUSTRIAL MEDICINE

Beryllium Granulomatosis Complicated by Tuberculosis: Report of a Case Treated with ACTH.

DOBSON, R. L., WEAVER, J. C. AND LEWIS, L.: ANN. INT. MED., 38: 312, 1953.

That physicians should consider beryllium among possible etiologies of obscure pulmonary disorders even when the occupational aspects are not obvious, is indicated by this article. In it the authors report in detail a fatal case of chronic granulomatosis of the lungs due to beryllium, complicated by tuberculosis.

The case is that of a 37 year old scientist engaged in research in a radiation laboratory, who took ill in the summer of 1945. At that time it was just beginning to be acknowledged that beryllium can induce serious pulmonary disease. The physicians who first attempted diagnosis considered the possibility of toxicity from radioactive materials. It was not until 5 years after the onset of illness and 8 years after the event that a history of brief exposure to beryllium was obtained. The case was then found in retrospect to follow in many respects the pattern of chronic beryllium granulomatosis.

Details are given regarding the course of the disease which during the latter stages was adversely affected by the unusual complication of an intercurrent tuberculous infection. By April, 1950 the patient was considered moribund. Cortisone therapy was instituted; after a short trial, it was replaced by ACTH. The initial response was dramatic. The ACTH produced a definite clinical remission, with improvement of objective as well as subjective manifestations of pulmonary disease. Until the spread of tuberculosis vitiated its beneficial effects, ACTH resulted in improvement of respiratory function as well as gain of weight and strength. The gradual deterioration which followed was considered attributable to progressive pulmonary tuberculosis and chronic hypoxia from respiratory insufficiency.

The pathologic observations indicate that pulmonary reactions to beryllium and to the products of the tubercle bacillus may become indistinguishable when the two diseases are superimposed. The presence of significant amounts of beryllium in tissue samples obtained at autopsy, demonstrate the relationship between the seemingly insignificant exposure of 8 years before and the patient's illness and death.

In the authors' opinion, a detailed history of employment, including the names of materials handled and the processes involved in the job, may be the only means of establishing an essential link in the chain of diagnostic evidence.

MARGARET H. WILTON

New Approaches to the Health Hazard Evaluation of Pesticides.

KINGSLEY, K.: ARCH. INDUST. HYG., 8: 70, 1953.

The active development of organic pesticides following World War II presented a new problem in public health as these compounds were found to be more potentially toxic to man than the inorganic chemicals previously employed. Lack of knowledge with regard to the mode of action of many of these agents has made it impossible to clearly evaluate them in their present day use. In this article the author indicates aspects of pesticide hazard evaluation which need more detailed study before new products are introduced to the market and discusses three new approaches as exemplified by experimental work carried out in the Occupational Health Laboratory of the Department of National Health and Welfare. These are:

1. *Toxicity of trade formulations versus pure compounds.*—Investigation was conducted in 1950 into the toxicity of trade formulations of aldrin and dieldrin. It was found that the value for the pure compound, which was not the material incorporated into trade formulations, was misleading. The findings demonstrate the importance of determining the toxicity of actual trade formulations in order to take into account toxicity contributions by impurities and associated materials.

2. *Difference between laboratory toxicity and hazard.*—There has been a lack of understanding of the factors which differentiate the toxicity for laboratory animals from the hazard to the exposed human. The health hazard evaluation of pesticides is a combination of toxicity and level of application. Field study of environmental exposure, clinical aspects and biochemical response in the subclinical range would appear to be the soundest approach. Such a study was carried out in the St. Hilaire orchards of Quebec to determine the hazard from parathion during spraying operations.

3. *Biochemical investigations of new pesticides.*—Study of biochemical response to new insecticides should be instituted as it is from such knowledge that clinically valuable tests and antagonizing agents may be developed and available for the health protection of exposed persons. Reference is made to some preliminary work which has been done along this line.

MARGARET H. WILTON

Canad. M. A. J.
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FORTHCOMING MEETINGS

CANADA

ONTARIO MEDICAL ASSOCIATION, Annual Meeting, Toronto, Ont. (Dr. Glenn Sawyer, 244 St. George Street, Toronto 5, Ont.) May 10-14, 1954.

CANADIAN UROLOGICAL ASSOCIATION, Annual Meeting, Thousand Island Club, Ont. (Secretary, Dr. S. A. MacDonald, 1414 Drummond St., Montreal, Que.) June 3-5, 1954.

INTERNATIONAL CONGRESS OF PSYCHOLOGY, Montreal, Que. (Prof. H. S. Langfeld, International Union of Scientific Psychology, Eno Hall, Princeton University, Princeton, N.J.) June 7-12, 1954.

SOCIETY OF OBSTETRICIANS AND GYNÆCOLOGISTS OF CANADA, Annual Meeting, Harrison Hot Springs, B.C. (Dr. R. B. Meiklejohn, Secretary, Toronto Western Hospital, Toronto, Ont.) June 10-13, 1954.

CANADIAN MEDICAL ASSOCIATION, Annual Meeting, Vancouver, B.C. (Dr. T. C. Routley, General-Secretary, 244 St. George Street, Toronto 5, Ont.) June 14-18, 1954.

CANADIAN TUBERCULOSIS ASSOCIATION, Saint John, N.B. (Dr. G. J. Wherrett, Executive Secretary, Canadian Tuberculosis Association, 265 Elgin Street, Ottawa 4, Ont.) June 24-26, 1954.

INTERNATIONAL CONFERENCE ON GROUP PSYCHOTHERAPY, Toronto, Ont. (Dr. Wilfred C. Hulse, Chairman, International Committee on Group Psychotherapy, 110 West 96th Street, New York 25, N.Y.) August 12-19, 1954.

INTERNATIONAL CONGRESS ON CHILD PSYCHIATRY, University of Toronto, Toronto, Ont. (Miss Helen Speyer, Executive Officer, International Association for Child Psychiatry, 1790 Broadway, New York 19, N.Y.) August 13-14, 1954.

INTERNATIONAL CONGRESS ON MENTAL HEALTH, University of Toronto, Toronto, Ont. (The Executive Officer, Fifth International Congress on Mental Health, 111 St. George Street, Toronto, Ont.) August 14-21, 1954.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY, Montreal, Que. (Dr. G. Stuart Ramsey, Associate Secretary, Physical Sciences Centre, McGill University, Montreal 2, Que.) September 9-11, 1954.

INDUSTRIAL MEDICAL ASSOCIATION OF THE PROVINCE OF QUEBEC, AND THE INDUSTRIAL SECTION OF THE ONTARIO MEDICAL ASSOCIATION, Joint Meeting, Ottawa, Ont. (Dr. W. F. Prendergast, Secretary of the Section of Industrial Medicine, 22 Commercial Road, Leaside, Toronto 17, Ont.) September 23-25, 1954.

UNITED STATES

AMERICAN ORTHOPSYCHIATRIC ASSOCIATION, Annual Meeting, New York, N.Y. (Dr. Marion F. Langer, American Orthopsychiatric Association, 1790 Broadway, New York 19, N.Y.) March 11-13, 1954.

AMERICAN HEART ASSOCIATION, Annual Meeting, Chicago, Ill. (Dr. Charles D. Marple, Medical Director, American Heart Association, 44 East 23rd Street, New York 10, N.Y.) April 1-4, 1954.

ANNUAL SYMPOSIUM ON RECENT ADVANCES IN THE STUDY OF VENEREAL DISEASES, Washington, D.C. (Dr. James K. Shafer, Public Health Service, Washington 25, D.C.) April 29-30, 1954.

AMERICAN GOITER ASSOCIATION, Annual Meeting, Boston, Mass. (Dr. John C. McClintock, Corresponding and Recording Secretary, 149½ Washington Ave., Albany, N.Y.) April 29 - May 1, 1954.

CONFERENCE OF CATHOLIC SCHOOLS OF NURSING, Annual Meeting, Atlantic City, N.J. (Executive Office, 1438 South Grand Blvd., St. Louis 4, Mo.) May 15-16, 1954.

CATHOLIC HOSPITAL ASSOCIATION, Annual Convention, Atlantic City, N.J. (Executive Office, 1438 South Grand Blvd., St. Louis 4, Mo.) May 17-20, 1954.

AMERICAN TRUDEAU SOCIETY, Medical Section of the National Tuberculosis Association, Annual Meeting, Atlantic City, N.J. (Chairman of the Medical Sessions Committee, American Trudeau Society, 1790 Broadway, New York 19, N.Y.) May 17-21, 1954.

AMERICAN UROLOGICAL ASSOCIATION, Annual Meeting, New York, N.Y. (Dr. William P. Didusch, Executive Secretary, 1120 N. Charles Street, Baltimore 1, Md.) May 31-June 3, 1954.

AMERICAN GERIATRICS SOCIETY, Annual Meeting, San Francisco, Calif. (Dr. W. O. Thompson, President, 700 North Michigan Ave., Chicago 11, Ill.) June 17-19, 1954.

AMERICAN COLLEGE OF CHEST PHYSICIANS, Annual Meeting, San Francisco, Calif. (Dr. Edgar Mayer, Chairman of the Committee on Scientific Program, 850 Fifth Avenue, New York 21, N.Y.) June 17-20, 1954.

AMERICAN MEDICAL ASSOCIATION, Annual Meeting, San Francisco, Calif. (Dr. George F. Lull, 535 North Dearborn Street, Chicago 10, Ill.) June 21-25, 1954.

ANNUAL SYMPOSIUM ON TUBERCULOSIS AND OTHER CHRONIC PULMONARY DISEASES FOR GENERAL PRACTITIONERS, Saranac Lake, N.Y. (Dr. Richard P. Bellaire, P.O. Box 2, Saranac Lake, N.Y.) July 12-16, 1954.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY, New York, N.Y. (Dr. William L. Benedict, Secretary-General, 100 First Avenue Building, Rochester, Minn.) September 12-17, 1954.

WORLD CONGRESS OF CARDIOLOGY, Washington, D.C. (Dr. L. W. Gorham, Secretary-General, Second World Congress of Cardiology, 44 East 23rd Street, New York 10, N.Y.) September 12-17, 1954.

INTERNATIONAL ANÆSTHESIA RESEARCH SOCIETY, Annual Congress, Los Angeles, Calif. (Dr. T. H. Seldon, Chairman, 102-110 Second Avenue, S.W., Rochester, Minn.) October 4-7, 1954.

OTHER COUNTRIES

INTERNATIONAL CONGRESS OF INTERNATIONAL COLLEGE OF SURGEONS, Sao Paulo, Brazil. (Dr. Max Thorek, Secretary-General, 1516 Lake Shore Drive, Chicago, Ill.) April 26-May 2, 1954.

IRISH MEDICAL ASSOCIATION, Annual Meeting, Killarney, Ireland. (Dr. P. J. Delaney, Medical Secretary, 10, Fitzwilliam Place, Dublin) July 7-10, 1954.

INTERNATIONAL GERONTOLOGICAL CONGRESS, London and Oxford, England. (Prof. R. E. Tunbridge, President, Department of Medicine, The University, Leeds, England) July 12-22, 1954.

INTERNATIONAL CONGRESS ON THROMBOSIS AND EMBOLISM, Basle, Switzerland. (Dr. W. Merz, Hon. Secretary, Gynæcological Clinic, University of Basle, Switzerland) July 15-19, 1954.

INTERNATIONAL CONGRESS FOR PSYCHOTHERAPY, Zürich, Switzerland. (Dr. H. K. Fierz, Secretary General, Theaterstrasse 12, Zürich 1, Switzerland) July 21-24, 1954.

INTERNATIONAL CANCER CONGRESS, Sao Paulo, Brazil. (Prof. A. Prudente, 171 rua Benjamin Constante, Sao Paulo, Brazil) July 23-29, 1954.

INTERNATIONAL CONGRESS ON OBSTETRICS AND GYNÆCOLOGY, Geneva, Switzerland. (Dr. H. de Watteville, President, Maternité Hôpital Cantonal, Geneva, Switzerland) July 26-31, 1954.

INTERNATIONAL CONGRESS OF INTERNAL MEDICINE, Stockholm, Sweden. (Secretariat of the Third International Congress of International Medicine, Karolinska sjukhuset, Stockholm 60, Sweden) September 15-18, 1954.

CONFERENCE OF THE INTERNATIONAL UNION AGAINST TUBERCULOSIS, Madrid, Spain. (Prof. Alix y Alix, Escuela di Tisiologia, Ciudad Universitaria, Madrid, Spain) September 26 - October 2, 1954.

PAN-PACIFIC SURGICAL ASSOCIATION, Congress, Honolulu, Hawaii. (Dr. F. J. Pinkerton, Director General, Pan-Pacific Surgical Association, Young Building, Honolulu, Hawaii) October 7-8, 1954.

JAPAN MEDICAL CONGRESS, Kyoto, Japan. Dr. M. Goto, Secretary General, University Hospital, Medical Faculty of Kyoto University, Kyoto, Japan) April 1-5, 1955.

NEWS ITEMS

BRITISH COLUMBIA

The Fifth Annual Meeting of the B.C. Division of the Canadian Arthritis and Rheumatism Society was held on Monday, January 18, in the Devonshire Hotel in Vancouver. The report of Mr. William Murphy, President, stated that about 45,000 treatments were given in 1953. The rehabilitation department (the first in Canada) has had an occupational therapist added to its staff. Centres have been opened for treatment during 1953 at Abbotsford, Alberni, Chilliwack and Courtney. There are five new mobile clinics, and a travelling consultant service has been established to visit patients all over B.C. Mr. Brenton S. Brown was elected President for the new year.

Dr. C. E. Robinson, former medical director of the C.A.R.S. has resigned, and is succeeded by Dr. R. W. Lamont-Havers. Dr. Harold S. Robinson has been appointed full time Director of Medical Research. Dr. Stewart Murray, Chief Medical Health Officer of the Metropolitan Health Department of Vancouver, has been appointed Vice-president of the American Public Health Association.

Dr. Kenneth E. Evelyn of Montreal has been appointed Director of the B.C. Medical Research Institute. In addition he will occupy the position of Reserve Professor in the Department of Medicine of the University of British Columbia. Dr. Evelyn was associate professor of medicine at McGill and Director of the Research Institute of Biophysics.

The new 52-bed Holy Trinity Hospital for chronic and convalescing patients, at Sixty-second Avenue and Argyle will open some time in March. It will be operated by the Sisters of Providence of St. Vincent de Paul.

The Provincial Biopsy Service, established by the Provincial Health Department in 1949, is doing a land-office business—13,059 specimens were examined in the last twelve months alone. This was designed to be chiefly a cancer service, to examine tissue suspected of malignancy, but actually it has spread to include a good deal of other examinations, and the Department is very generous in its attitude towards the medical profession in this regard.

The two new wings of the Western Rehabilitation Society on 27th Ave. West in Vancouver will be opened formally on March 11, and a special Open House will be held for all members of the Medical profession from 11.00 a.m. to 4 p.m.

Dr. Edith Whetnall, F.R.C.S. (Eng.), was a recent visitor to Vancouver, where she met many organizations and individuals, who are interested in deaf children and their rehabilitation. She is Consultant in Otology to the London (Eng.) County Council, and is a director of the Audiology Unit of the Royal Ear, Nose and Throat Hospital, London. Her visit was a very stimulating experience to all who met her, and she gave generously of time and effort, in explaining her methods and techniques of treatment. J. H. MACDERMOT

MANITOBA

The Board of Governors of the University of Manitoba has submitted to the provincial cabinet a request for a sum, reported to be in excess of $3,000,000 for renovation of the Medical College. The program of expenditure is to be spread over a period of years. The first year extension will call for at least $750,000 for a new library. Some of the older buildings are due for replacement and a large auditorium is also under consideration. If the plan is approved, the quota of students admitted each year can be increased.

The latest issue of the *Manitoba Medical Students Journal* is devoted entirely to poliomyelitis with articles by Dean L. G. Bell; Dr. J. A. Hildes, Superintendent of Municipal Hospitals who was recently voted Winnipeg Citizen of the Year; Dr. J. D. Adamson, head of the Medical Advisory Board; Dr. Roper Cadam, assistant health officer of Winnipeg; Dr. A. J. W. Alcock, Dr. J. C. Wilt, Dr. J. B. R. Cosgrove, Dr. M. H. L. Desmarais, and Mr. M. Rosenberg.

Winnipeg ratepayers will shortly be asked to vote on a referendum on the question of whether the city should guarantee debentures for $2,000,000 issued by the Winnipeg General Hospital.

City of Winnipeg officials, acting on the report of a financial firm, have requested the province to approve a health unit for Winnipeg. If approved, the province may pay about two-thirds of the cost of the health unit. Brandon and St. Boniface now have provincial health units.

Wallace A. McAlpine, F.R.C.S.(Eng.), F.R.C.S.(Edin.) is now associated with the Manitoba Clinic and will confine his practice to thoracic and cardiac surgery.

J. Reid Taylor, M.D., is associated with Dr. H. D. Morse and Dr. C. B. Stewart of the Winnipeg Clinic in the practice of urology. ROSS MITCHELL

NEW BRUNSWICK

Dr. O. B. Evans has resigned as Chief of Service, Department of Surgery of the Saint John General Hospital staff, after thirty years of continuous service in the Department of Surgery. He has been succeeded as Chief of Service by Dr. R. M. Pendrigh.

Dr. Edgar M. Copp died in a nursing home in Hampton, N.B. on January 19. Dr. Copp was born in Jolicure; was educated in local schools and qualified as a teacher following which he taught school for several years. He graduated from Jefferson Medical School and practised medicine in Sackville for more than fifty years. He was a member of the United Church and a Mason.

Dr. E. A. Petrie, radiologist at Saint Joseph's Hospital in Saint John, has been granted a fellowship in the American College of Radiologists. Dr. Petrie has given a great deal of unselfish service in the Radiological Society of North America of which he was a vice-president and, in the Canadian Association of Radiologists of which he has been president; and he headed a committee of radiologists which co-operated with the x-ray technicians' society in preparing a curriculum and setting standards for registration of technicians.

A. S. KIRKLAND

ONTARIO

Essex County Medical Society is starting a Benefit Fund. Each member under sixty-five years of age is to contribute ten dollars for two years. This will form a capital of $4,000. The capital is to be maintained at this level by an annual taxation when necessary. Any member of the Society who has paid his dues and who has an illness of six months or longer will be eligible for benefits.

At the second postgraduate course in Diabetes and Basic Metabolic Problems held at Rochester, Minn. in January the speakers from Toronto were Dr. C. H. Best whose topic was "About the Pancreas"; Dr. A. L. Chute who spoke on "Experiences with NPN Insulin in Childhood Diabetes" and Dr. G. A. Wrenshall who spoke on "The Hyperglycæmic Glycogenolytic Factor of Pancreas".

The Toronto General Hospital Alumnæ Association formally presented the Jean I. Gunn Memorial Library in the new School of Nursing to the University of Toronto. Dr. Sydney Smith accepted the gift on behalf of the University. The late Miss Gunn was an international leader in the field of nursing and superintendent of nurses at Toronto General Hospital for more than twenty-five years. Dominating the Library which has been completely and handsomely furnished by the alumnæ who raised $15,000 for the purpose is a portrait of Miss Gunn by Cleeve Horne who captured the blending of strength and humaneness which characterized her.

Dr. R. F. Farquarson has been made an Honorary Citizen of the State of Texas.

Dr. R. M. Janes has been elected an honorary member of the Society of Thoracic Surgeons of Great Britain and Ireland. Dr. Janes recently addressed the Los Angeles Surgical Society on "Postoperative Complications" and at the same meeting was made an Honorary Member of the Los Angeles Surgical Society, a week later he spoke to the Hollywood Academy of Medicine on "The Surgical Management of Lesions of the Breast".

Dr. E. V. Kalyani, honorary obstetrician and gynæcologist at the Government Hospital for Women and Children in Madras, and Dr. P. M. Naidu, professor of obstetrics and gynæcology in Hyderabad, India, have been visiting in the clinics of the department of obstetrics and gynæcology, University of Toronto. While they were here they were shown the work in the Cancer Clinic and the gynæcological operative procedures carried out in the university hospitals of Toronto.

About a hundred members attended the annual meeting of the Ontario Psychological Association held in Ottawa in January. The president of the Association is Dr. G. H. Turner, Department of Psychology, University of Western Ontario. Symposia were held on Child Psychology, Personnel Psychology, Training in Human Relations and on Psychotherapy. Dr. Fredrick C. Thorne, professor of Psychiatry, University of Vermont gave an address on Principles of Psychological Examination.

Dr. Jessie Gray, chief of the surgical staff at Women's College Hospital received an Elizabeth Blackwell citation in New York for her work in surgery. The ceremony was connected with the centennial celebrations of the New York Infirmary, founded by Dr. Elizabeth Blackwell. The hospital is staffed entirely by women.

A $90,500 federal grant has been allocated for extension of the Sudbury General Hospital. This grant will be used to build accommodation for 38 active treatment patients and 35 psychiatric patients. This grant is the fourth made to the Sudbury hospital and brings the total to $292,600.

Dr. H. W. Kosterlitz, University of Aberdeen and the Harvard Medical School, addressed the Physiological Society of the University of Toronto on The Liver in Pregnancy.

LILLIAN A. CHASE

QUEBEC

On January 25, the Quebec Legislative Assembly unanimously approved a bill authorizing the Cabinet to set up a committee to report on the state of hospitals in the Province. Health Minister Paquette informs us that three men will be appointed to survey the hospitals, study the possibility of building convalescent homes and inquire generally into hospital systems. Since 1945, the Quebec Government has built 91 hospitals and sanatoria in order to meet urgent needs. It now wants to get an overall view of the situation, especially the need for convalescent homes. The chief problem today is hospital costs and it is hoped that this Committee will look into this problem.

On January 27 Dr. Marc Trudel, president of the College of Physicians and Surgeons of Quebec, presented to the Quebec Royal Commission on Constitutional Problems a 48-page brief on behalf of the College. This brief stressed opposition to the 1948 National Health Program and advocated a contributory provincial plan that would cover hospitalization and medical care. Demands for national health stem from the fact that most existing prepaid plans do not provide hospital and medical coverage. There are no provisions in these plans for treatment at home or in the doctor's office.

The brief urged that the province maintain "the traditional practice of medicine", the free choice of the doctor by the patient and vice versa. Neither provincial authorities nor the College will recognize a prepaid health plan which does not cover the following conditions: a provincial charter, hospital coverage, home and office medical services, and medical services of the highest quality.

The 17th Annual Meeting of the Canadian Association of Radiologists was held in January at the Chateau Frontenac in Quebec City. Your reporter did not have

the good fortune to enjoy this gathering of some 200 delegates, but from all reports it was most successful. The Gordon Richards Memorial Lecture was delivered by Dr. Carleton B. Pierce, Royal Victoria Hospital. Dr. J. W. McKay, professor of radiology at McGill and chief radiologist at the Montreal General Hospital, was elected president of the Association.

The annual lecture in memory of Dr. H. B. Cushing, McGill's first professor of pædiatrics, and sponsored by the Nu Sigma Nu medical fraternity and the Medical Undergraduate Society, was given by Dr. Max Thorek at the Children's Memorial Hospital on January 15. Dr. Thorek, who is secretary-general of the international College of Surgeons and surgeon-in-chief at the American Hospital in Chicago, spoke about emergency resuscitation following heart stoppage. He stressed the need for speedy and efficient team work between surgeon and anæsthetist before, during and after operations to prevent and deal with such crises. Dr. Thorek also showed a motion film recording the case of a young woman whose heart stopped during an operation. Dr. Thorek massaged the heart and restored its action after 19 minutes.

On January 22nd the medical staff of the Children's Memorial Hospital presented its first "quiz program". This meeting was under the chairmanship of Dr. Dudley Ross and some 100 physicians were in attendance. Among subjects discussed were: when should children have their tonsils removed, latest treatment for burns and scalds in children, and the use of tonics.

At a meeting of the Montreal Neurological Society held at the Montreal General Hospital on January 13, Drs. Miller Fisher, Harold Elliott, W. F. T. Tatlow and H. H. Jasper discussed the problem of unconsciousness. They reviewed studies done on damaged brains and listed what is known of the causes of unconsciousness in relation to specific portions of the brain.

A. H. NEUFELD

SASKATCHEWAN

The Regina and District Medical Society has inaugurated a series of panel discussions as a public service project for the benefit of the people of that district. Their first presentation was held in conjunction with the Saskatchewan Council for Crippled Children and Adults, and was a very definite success, as indicated by the very large attendance and the quality of the program provided.

Dr. G. W. FitzGerald, President of the Regina branch of the Council for Crippled Children and Adults in introducing the Panel indicated that it was hoped that the work of the Society would be fostered and aided by bringing this type of information to the public. "Doctors are becoming increasingly tired of trying to cope silently with prepaid misinformation obtained from articles in popular magazines. Too often these contain medical half-truths that mislead the public. Despite spectacular and great advances in medical science during this century, quackery still thrives on the silence of the medical profession. While it is not possible to cure every disease, the medical profession feels that no one should be deprived by quackery, or misinformation, or through gross ignorance of the best that modern medicine can bring", he said.

The members of the panel consisted of Drs. A. J. Longmore (moderator), George Walton, Mitchell Finklestein, M. G. Israels, Harold Graham, H. J. Spooner, Barry Duncan and James Stephens. The panel discussed the subject of poliomyelitis and then opened the meeting to a question and answer period.

Saskatchewan had the second highest incidence of polio in the Province's history last year. While the figures reported are subject to revision, it would appear as if there were 1,086 cases of the disease as compared to 1,223 in 1952. There were 70 polio deaths among these reported cases (89 in 1952). In comparison with last year's epidemic the southern part of the Province bore the brunt of the illness. The 1952 epidemic seemed to settle in the northern areas.

In connection with the above figures, it might be noted that Saskatchewan was the only Western Province where the polio outbreak was less severe than the 1952 one. Alberta had some 1,300 cases and 98 deaths, and Manitoba had more than 2,300 cases, the per capita incidence apparently exceeding all known Canadian records.

Saskatchewan also reversed the national trend towards a higher percentage of paralytic cases. Of the 1,086 reported, 27% were said to have some paralysis. 50% of last year's cases were in persons 15 years of age or over.

Dr. Irwin M. Hilliard, B.A., M.D., F.R.C.P.[C], has been appointed Professor and Head of Medicine at the University of Saskatchewan. A graduate of the 1936 class in Toronto, Dr. Hilliard spent three years in medicine at the Toronto General Hospital and at West China Union University before taking his post at the Canadian Mission Hospital at Fowling, Szechuan. There he had a wide experience, extending from rural medical practice to the responsibilities of physician-in-chief to a busy hospital with an active intern and nursing training program. In 1945, following the political upheaval, Dr. Hilliard returned to Canada to engage in Clinical research and department activities in medicine at the Toronto General Hospital. His interests there, and subsequently at the Toronto Western Hospital have included studies in cardiology and in pulmonary function. Last year he served as chairman of the Section of Medicine of the Academy of Medicine in Toronto and as chairman of the Central Program Committee of the Canadian Medical Association. He will take up his duties in Saskatoon in the summer of 1954.

Announcement is made by Dean J. W. Macleod of awards for travel for Dr. Albert B. Brown, Baltimore, recently appointed Professor of Obstetrics and Gynæcology. The Commonwealth Fund of New York and the Nuffield Foundation of England will provide Dr. Brown with an opportunity to visit medical centres in America and Europe during the next six months. Dr. Brown, a graduate of Queen's University, Kingston, has been attached to the Johns Hopkins Hospital during the past six years.

G. W. PEACOCK

NEWS OF THE MEDICAL SERVICES

Canadian Armed Forces

The Royal Canadian Navy announced the promotions of three medical officers to the rank of Surgeon Commander on January 1, 1954. They were: Acting Surgeon Commander M. H. Little, Head of Department of Anæsthesiology at R.C.N. Hospital, Halifax, N.S., confirmed in rank of Surgeon Commander, R.C.N.; Surgeon Lieutenant Commander R. F. Hand, Otolaryngologist at R.C.N. Hospital, Halifax, N.S., to be Surgeon Commander, R.C.N.; Surgeon Lieutenant Commander J. W. Green, Principal Medical Officer of H.M.C.S. *Ontario*, to be Acting Surgeon Commander, R.C.N. Other promotions of interest included two medical officers who have just returned from service in the Korean theatre. Both Surgeon Lieutenant W. C. Wood, R.C.N. of H.M.C.S. *Iroquois* and Surgeon Lieutenant R. B. Irwin, R.C.N. of H.M.C.S. *Athabaskan* became Surgeon Lieutenant Commanders.

Surgeon Lieutenant G. R. Holmes, R.C.N., who recently completed a Senior Internship at Victoria General Hospital, Halifax, N.S., was promoted to Surgeon Lieutenant Commander.

Colonel J. E. Andrew, E.D., and Colonel G. L. M. Smith, C.B.E., of the staff of the Director General of Medical Services, attended a short course on medical care of atomic casualties from January 6 to 16, 1954 at the Army Medical Services Graduates School, Walter Reed Army Hospital, Washington.

Major J. S. Hitsman, M.B.E., has been promoted to the rank of Lieut.-Col. and has been appointed to command Fort Churchill Military Hospital, Fort Churchill, Manitoba.

The following officers of the Directorate of Medical Services (Air) visited R.C.A.F. units in the U.K. and the Continent during the month of February: Group Captain G. D. Caldbick; Squadron Leader J. D. Duncan; Squadron Leader F. M. Oakes.

The 24th Meeting of the Advisory Medical Committee to the R.C.A.F. was held in Ottawa February 20, 21, 1954.

NEWS AND NOTES

THE J. A. STEWART DORRANCE MEMORIAL AWARDS

J. A. Stewart Dorrance, M.D.C.M. (Queen's 1947) died at the age of thirty before he had even begun private practice. Yet, in his short life some glowing warmth of sympathy kindness and gentleness set him apart. A friend, who had been helped by his goodness, established awards, in the Medical Faculty under conditions to be determined by his father and mother.

Professor and Mrs. Dorrance decided on two awards to be given at the end of the fourth and fifth years, tenable in the following year to students who seem to their classmates and teachers most nearly to possess those qualities which made their son a joy to his parents, a comfort to his patients and a gracious influence on all who knew him.

The selections in each year will be made by a committee appointed by the Dean who will be guided by a ballot by members of the fourth and of the fifth years respectively.

The amount of the awards in 1954 will be $200. each.

NUTRITIONAL EDUCATION

In old age, as at other ages, education is the most reliable way of assuring good nutrition. For older people who care for themselves, nutritional education cannot be stressed too much. Their nutritional knowledge lags far behind that of their younger contemporaries. Emphasis should be placed on practical matters, but the reasons behind the nutritional advice should be explained. The following may serve as reminders of important points to be covered.

1. A good diet is just as important in old age as in youth.
2. Overeating is more dangerous for the old than for the young.
3. Older people should be sparing in the use of all fats and oils and should avoid cooking in fat.
4. The simplest way to assure adequacy of proteins, vitamins, and minerals is to use a varied diet made up of natural foods with a minimum of processing.
5. Cooking in large quantities of water is certain to result in losses of vitamins and minerals.
6. Liberal use of leafy and root vegetables, fruits, and coarse cereals will help control constipation. Failure to have a regular bowel movement every day is not constipation.
7. Bread enriched with milk solids and vitamins is desirable.
8. There should be an abundant fluid intake, and this may include moderate amounts of coffee, tea, and alcoholic beverages. The latter are best taken with, or immediately before, meals.
9. Meat, fish, or eggs every day should be the rule.
10. Special food concentrates and nostrums are seldom necessary. Peculiar and fancy diets should be avoided.
11. Moderate limitation in the use of table or cooking salt is wise.—Ancel Keys: *Pub. Health Rep.*, 67: 484, 1952.

MOSQUITO CONTROL

During the past year, there have been indications from several scattered areas in the world that some species of *Anopheles* are developing resistance to DDT. Some evidence has been developed by workers of the Tennessee Valley Authority that *Anopheles quadrimaculatus* may be developing resistance to DDT in some localities in which DDT has been used continuously for more than 5 years. However, in general this species does not appear to have developed resistance to DDT to a degree which would significantly affect control operations.

The only known mosquito vector of disease which appears definitely to have developed a high degree of resistance to insecticides in the United States is *Culex tarsalis* in California. Observations by the Bureau of Entomology and Plant Quarantine of the United States Department of Agriculture indicate that in some localities this species has developed varying degrees of resistance to a number of chlorinated hydrocarbons, including DDT, toxaphene, lindane, aldrin, and heptachlor applied as space sprays. Although no specific recommendations can be made for control of the resistant strains of this species, DDT-DMC combinations, and perhaps other DDT-synergist combinations as they become available, may be used on a field trial basis. Combinations of DDT with DMC (*p*-dichlorodiphenyl methyl carbinol) at ratios from 5:1 to 20:1 have been effective as space sprays against field strains of DDT-resistant houseflies.—*Pub. Health Rep.*, 67: 455, 1952.

PUBLIC RELATIONS RESOLUTIONS—1954 STYLE, BUT ALWAYS GOOD

Avoid the evils of professional jealousy and unwarranted criticism of one M.D. by another. Such actions harm all doctors.

Increase individual and society community participation.

Increase publicity of grievance committees; provide "teeth" to discipline the mercenary and unethical M.D.'s who damage the reputation of all.

Increase participation in an Emergency Call System to keep it smooth working; publicize it.

Encourage the voluntary health insurance plans—the good ones!

Encourage and back up your society in a plan guaranteeing medical service to all regardless of ability to pay.

Encourage the establishment of active tissue committees in your hospitals to help combat fee splitting. (*Saturday Evening Post*, January, 16, 1954.)

Present a united front, supporting your society on medical issues.

Utilize all outlets (newspapers, radio, TV, speaker's bureaus, forums, etc.) to tell medicine's *positive* story.

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(Continued on page 84 of the advertising section)

BOOK REVIEWS

CORTISONE AND ACTH IN CLINICAL PRACTICE

Edited by W. S. C. Copeman, Physician, Department of Rheumatism, West London Hospital, and to Arthur Stanley Institute for Rheumatic Diseases of the Middlesex Hospital, London. 225 pp. illust. $5.00. Butterworth & Co. (Canada) Ltd., Toronto 6, 1953.

This easily read volume contains much useful information on the actions and uses of cortisone and ACTH. As its general editor is a rheumatologist it is not surprising that the largest section is devoted to the rheumatic disorders. The other chapters are written by various London physicians and describe the treatment of various inflammatory and allergic disorders of the eye, skin and the respiratory system, and the endocrine gland diseases. There is also a summary of what is known about their physiological and pharmacological actions.

Since it is now four years since the introduction of these hormones into clinical therapeutics, a review such as this is timely. However, by the time publication is achieved, there will be inevitably some facts that have become outdated. But it is surprising, on reflection, how little new knowledge has been added to this subject after the first few exciting months. Although they have now become more readily available, too much so perhaps, and hydrocortisone has been added, no new advance in understanding the basic mechanisms underlying their action has been made.

The indications for and against their exhibition have become more clearly established in most instances, but the really long term effects are only now beginning to become manifest. For instance, it is being appreciated—and this is not sufficiently stressed in the book—that a patient taking cortisone or who has recently taken it, may rapidly and unexpectedly go into acute adrenal failure when subjected to the stress of an infection or operative procedure, with perhaps a fatal outcome. Also in arthritis, further permanent damage may be caused by weight-bearing on hips and knees made pain-free by cortisone.

The otherwise good quality of this volume is somewhat marred by the rather pathetic and doubtful claim by the editor that he was the first to use cortisone in Britain. At the same time, he neglects to give credit to other original work in this field by some of his British colleagues. However, this should not detract from the fact that this is a book worth reading, and should be especially valuable to the general practitioner who wonders about the advisability of the use of cortisone and ACTH in his clinical practice.

THE PHYSICIAN IN ATOMIC DEFENSE

T. P. Sears, Associate Clinical Professor of Medicine, University of Colorado School of Medicine; Chief of Medical Service, Veterans' Administration Hospital, Denver. 308 pp. illust. $6.00. The Year Book Publishers Inc., Chicago, Ill., Burns & MacEachern, Toronto 2, 1953.

This is a timely book about a subject of transcendent importance. Recognizing the key rôle of the medical profession in regard to Atomic Defense, Dr. Sears has presented in a lucid, logical manner the technical information which is essential to that rôle. Here the physician will find expounded in a readily understandable manner the mysteries of atomic structure, isotopes, radioactivity, nuclear fission, the chain reaction and the atomic bomb. The effects of atomic bombing are graphically portrayed, and the Civil Defense organization required to cope with the problem is clearly outlined. Although written by an American for use in the U.S.A., almost everything that is said about Civil Defense organization is, fortunately, applicable in Canada, since the programs of the two countries have been closely synchronized. This book will serve as a useful reference for those physicians who play active rôles in Civil Defense; it should, at the same time, serve as an authoritative guide to local medical societies or hospital groups who recognize the need for indoctrination in this vital matter. For those not primarily interested in Civil Defense this book will serve as a helpful introduction to nuclear physics and its impact upon medical science.

AN APPROACH TO CLINICAL SURGERY

G. H. C. Ovens, Professor of Surgery, University College of the West Indies. 309 pp. illust. $4.50. J. & A. Churchill Ltd., London W.1; British Book Service (Canada) Ltd., Toronto, 1953.

This is a most useful book designed to be a guide to the medical student in his transition from the laboratory to the ward, the test-tube to the patient. It is concise, readable and surprisingly inexpensive in this day of the shrunken dollar. The introduction contains much good advice to the student on his attitude to patients and his work. There are pearls on the importance of practical experience, the ubiquity of teachers if stimulated by student interest and the value of the written report and progress note. The book is divided into two parts. The first deals with general principles. The second with the history-taking and physical examination of surgical cases. The section on principles deals with asepsis, inflammation, wounds, hæmorrhage, shock, infection, operations and dressings. Each of these is neatly dealt with in about 10 pages. The chapter on operations well illustrates the author's approach. There are paragraphs on the place of operations in the therapeutic armamentarium; skin preparations; positions on the operating table; incisions; suture methods; and indications for drainage. Finally, there is a useful glossary to simplify operation nomenclature.

In part two, after reviewing the important features of obtaining and recording a good history, common manifestations of surgical examination are reviewed. There are chapters on tumours and swellings, the surgical abdomen; the anus and rectum, the urinary system, ulcers, the tongue, the lymphatics, the breast, acute head injuries, fractures, joints, and peripheral vascular disease. These are well illustrated with line drawings. To one who has laboured through large texts seeking, but not always finding, the information here so succinctly expressed, this book is a pleasant surprise. I recommend it warmly to students and student teachers as an excellent introduction to practical surgery.

SACRAL NERVE-ROOT CYSTS

I. M. Tarlov, Professor of Neurology and Neurosurgery, New York Medical College, New York City. 134 pp. illust. $7.25. Charles C. Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1953.

This is an excellent monograph, describing a previously unrecognized cause for sciatic and perineal pain, urinary disturbances, and sensory changes in the sacral skin segments.

The author had found in anatomical studies that perineurial cysts are relatively common in the sacral region, attached to dorsal nerve roots close to the ganglion, and may also occur at higher levels. They may be single or multiple. While they do not necessarily produce symptoms, he now reports in detail four personal cases, in which a perineurial cyst was found and removed, with relief of symptoms. Six similar cases from the recent neurosurgical literature are also described.

From the history and neurological findings one is most likely to suspect a protruded intervertebral disc. Myelographic findings are not characteristic and may be

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normal. The cyst may only be found on exploration of the affected dorsal root close to its ganglion, following removal of the posterior sacral arch.

The author gives a detailed and clear description of the normal anatomy and histology of the filum and cauda equina, based on his earlier studies, and well-illustrated by drawings and photographs. Pathology and pathogenesis are also fully discussed.

This monograph should prove of interest to neurologists, neurosurgeons and orthopædic specialists.

ON BURNS

Edited by N. A. Womack. 178 pp. $6.00. Charles C. Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1953.

This book is divided into thirteen chapters and the discussion includes the treatment of burn shock, enzymatic debridement plasma substitutes, skin grafting of acute burns, late contractures, response of the adrenal gland, and nutrition.

The avoidance of over dosage with morphine to relieve pain is stressed. Moderate doses, graded according to the patient's condition and reinforced with a barbiturate were thought to be more effective in relieving both pain and apprehension by not interfering with the evaluation of the degree of shock present. The comparative advantages and disadvantages of the open method and closed method of treating acute burns is discussed. It is apparent that there is a place for each method in the treatment of burns and that complete adherence to one method or the other which is advocated by some surgeons is neither necessary nor desirable. The use of enzymatic debriding agents is described and rapid debridement of burned areas can be accomplished with some of these. However, the problem is complex and it is important that such preparations should be devoid of toxic or antigenic effects when applied to burns in man. The treatment of burn shock is thoroughly discussed and the advantages of plasma substitutes of various types in the treatment of mass casualties is pointed out, since supplies of blood and plasma might not be adequate. The rôle of Z-plastys, skin grafts and pedicle flaps in the treatment of late burn contractures is well described as a prerequisite to final rehabilitation of the patient. The use of cortisone and ACTH is discussed and the few indications in which these hormones may be of use are described. Finally the importance of adequate nutrition throughout the course of treatment is described.

This volume will be of interest to those surgeons whose practice includes the treatment of thermal burns and to others interested in the Civil Defense Program. The inclusion of a bibliography would have been a desirable addition.

GASTRIC CANCER

A. H. Iason, Attending Surgeon, Adelphi Hospital; Director of Surgery, Brooklyn Hospital for the Aged. 316 pp. illust. $8.25. Grune & Stratton, New York; The Ryerson Press, Toronto, 1953.

This book in which the author sets out to "summarize succinctly the cumulative literature concerning gastric cancer" is disappointing in many respects. At first glance, the excellence of the illustrations and reproductions gives rise to the hope that in the text will be found a clear-cut presentation of the facts and of the problems of the disease. This hope is short-lived for it is soon apparent that the author intends to confine himself to a summary of the literature without attempting to submit his own judgment of any of the controversial issues. The summaries on anatomy, pathology, etiology, symptomatology and treatment add very little to the material that is available in standard surgical textbooks.

ROENTGEN, RADIUM AND RADIOISOTOPE THERAPY

A. J. Delario, Member of the American College of Radiology, American Board of Radiology. 371 pp. illust. $8.25. Lea & Febiger, Philadelphia; The Macmillan Co. of Canada Ltd., Toronto 2, 1953.

The author has covered most of the aspects of therapeutic radiology in a comprehensive fashion. A very considerable portion of the text deals with the basic fundamentals of radiological physics and the reaction of normal and malignant cells to irradiation. On of the features is the coverage of radioactive isotopes. This new field of therapy holds great promise, and the author has summarized in admirable fashion the present trend towards the diagnostic and therapeutic uses of these radio-active elements. It should prove to be a very useful reference in this new field.

It is imperative that all physicians, and in particular, radiologists acquaint themselves with the effects of the atom bomb. The author has gone to some detail in describing the effects of an atom bomb explosion on the general population, and what procedures should be employed in such catastrophe. This discussion has been presented in a most interesting and stimulating fashion.

If there is a fault to find, it would be of the description and discussion of diseases amenable to radiation therapy. X-irradiation comprises the bulk of the work in most radiotherapeutic departments and there is no doubt that most of the procedures employed are of a stereotype fashion. Nonetheless, it would appear that a more detailed discussion of diseases amenable to radiation therapy, particularly in relation to the time and dosage factors employed would be advantageous. Considerable effort has been directed to the discussion of natural radioactive substances and this aspect of therapeutic radiology would be of value to all therapists.

This text should be received by physicians generally in a welcome manner. It is concise and contains abundant references throughout the volume. These advantages alone make it a worthy part of a library for both students and physicists and for those requiring information from a reference library.

SYNOVIAL FLUID CHANGES IN JOINT DISEASE

M. W. Ropes, Associate Physician, Massachusetts General Hospital, Assistant Clinical Professor of Medicine, Harvard Medical School; and W. Bauer, Chief of Medical Services, Massachusetts General Hospital. 150 pp. illust. $4.40. Harvard University Press, Cambridge, Massachusetts; S. J. Reginald Saunders & Co. Ltd., Toronto, 1953.

This book contains a large amount of factual data accumulated by the authors over a period of twenty years. Studies have been made of approximately 1,500 synovial fluids and the various characteristics recorded. Detailed description is given of the gross appearance of the fluid, the bacteriology and immunology, the content of total protein, fibrinogen, mucin and other non-electrolyte and electrolyte constituents. Considerable diagnostic value is placed on the number and character of the cells in the synovial fluid and upon the character of the mucin when precipitated with acetic acid.

The authors advise more frequent aspiration of joints containing fluid. This will prevent injury to the capsular tissue by distention and give the patient much relief from his discomfort. At times such measures may have considerable diagnostic value; several illustrative cases are presented.

These observations support and enlarge the conclusions of many recent writers that the synovial membrane is not a true membrane but a layer of modified and highly vascular connective tissue. Thus the synovial

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cavity is regarded as a connective tissue space and the synovial fluid is looked upon as tissue fluid with mucin (muco-polysaccharide) added by the connective tissue cells. In disease however, the nature of the synovial fluid may be much altered by such factors as changes in the permeability of the synovial membrane, the addition of the products of inflammation to the fluid, changes in the metabolic processes within the joint cavity and interference with the rate of absorption.

PITUITARY CHROMOPHOBE ADENOMAS

J. I. Nurnberger, Research Associate, The Institute of Living, and Assistant Clinical Professor of Medicine (Neurology), Yale University School of Medicine; and S. R. Korey, Associate Professor of Neurology, The School of Medicine, Western Reserve University. 282 pp. illust. $7.00. Springer Publishing Company, Inc., New York; Burns & MacEachern, Toronto 2, 1953.

This interesting monograph is called a clinical study of the sellar syndrome. The approach is wider than any one specialty and it succeeds in its aim of correlating the several medical disciplines necessary to the full understanding of this particular pathological manifestation of pituitary function. There are clear and concise remarks on the embryology, cytogenesis, anatomical relations, blood supply and innervation of the pituitary gland. The functional anatomy of the peri-sellar region includes a lucid account of our present knowledge of the differentiation of hypophyseal and hypothalamic function and their possible inter-relationships. The section on metabolism traces the development of the experimental and clinical observations in respect to the various endocrine functions and presents concise summaries after each specific function is reviewed in detail. The main contribution of this monograph is supplied by a detailed study of 117 patients with chromophobe adenomas. Initial symptomatology and neurologic findings are discussed including pertinent information in respect to visual symptoms, headache, extraocular nerve involvement, extra-sellar extensions, etc. The metabolic symptomatology is analyzed in respect to thyroid function; adrenal function studies reveals the interesting fact that 25% had symptomatic hypoadrenalism; gonadal function and carbohydrate metabolic activity are also fully dealt with. The inter-relation of endocrine and metabolic function and the influence of tumour size and many other miscellaneous observations are made. In differential diagnosis the value of air studies and lack of value of the E.E.G. is pointed out. Pathological studies and apparent subdivisions continue to be disappointing in correlation with clinical material. Therapy is discussed, with radio therapy as the treatment of choice with surgery as a secondary therapeutic measure. Substitution therapy is stressed. This monograph has an excellent bibliography and should find its way into the preferred literature of all physicians interested in this condition or its associated metabolic manifestations.

LECTURES ON THE SCIENTIFIC BASIS OF MEDICINE

Volume I, 396 pp. illust. Price 30s. The Athlone Press, London, W.C. 2, 1953.

This is the first annual volume based on a series of lectures arranged each winter by the British Postgraduate Medical Federation. Eighteen lectures of the 39 given during the winter of 1951-52 are included in this volume. Topics discussed range over a wide field and they are all excellent. Two of them, "Studies of Normal and Pathological Physiology of the Kidney" by Van Slyke and "Growth of the Human at the Time of Adolescence" by J. M. Tanner, are quite lengthy and should properly be considered as reviews rather than lectures. A most enjoyable and instructive volume.

THE PHARYNX—BASIC ASPECTS AND CLINICAL PROBLEMS

Edited by A. R. Hollender, Professor of Otolaryngology, Emeritus, University of Illinois College of Medicine; Attending Otolaryngologist and Chairman of the Service, Mount Sinai Hospital of Greater Miami. 560 pp. illust. $15.00. The Year Book Publishers Inc., Chicago; Burns & MacEachern, Toronto, 1953.

On picking up a 560 page volume at $15.00 entitled "The Pharynx" one would expect a concise, authoritative volume adhering closely to its subject. Unfortunately such is not the case and this book wanders into the mouth, to the salivary glands and a discussion of pædiatric problems. There are verbose, meaningless introductions to each chapter such as "The physiologic basis of medical practice is now recognized as the fundamental without which the pathologic basis would be deficient," "The physiologist has been lax in solving the functions of the pharynx," "Though the pharynx is a multistructured organ, it is not an isolated one," etc.

This is a book which might well have been left unpublished,—at best it could have been condensed to one-fourth and included as a chapter in an ear, nose and throat text.

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THERAPEUTICS IN INTERNAL MEDICINE

Edited by F. A. Kyser, Assistant Professor of Medicine, Northwestern University Medical School, Chicago; Attending Physician, Evanston Hospital, Evanston, Ill. 830 pp. Revised 2nd ed. $15.00. Paul B. Hoeber, New York 16, N.Y., 1953.

One could not wish for a better textbook of therapeutics than this, which brings one the practical experience of 84 contributors. Authoritative, concise and up-to-the-minute, it is so edited that it is easy to find the answer to one's immediate therapeutic problem. At a time when many new drugs are being advocated by the medical literature, detail men and medical advertising, it is reassuring to have the opinions of unbiased investigators at hand for guidance. The book is so written that whenever necessary current concepts of physiology, pathology and etiology are discussed to provide a rational basis for the suggested treatment. As a general rule these aspects of the various diseases are so well written that one is apt to find oneself thumbing through other pages than those relating solely to the problem of the moment. It is found to be most interesting to wander and renew one's acquaintance with rare and unusual syndromes or to review the signs of toxicity from certain drugs; to glance perhaps through industrial poisons or perhaps compare one's own time-tested diets with those throughout the book for new ideas.

There will be instructive and pleasurable reading for those who may purchase this book to guide themselves through today's ever enlarging field of therapeutics.

THE OBSTETRICAL FORCEPS

L. V. Dill, Head, Obstetrics and Gynæcology, Yater Clinic; Assistant Professor, Obstetrics and Gynæcology, Georgetown University School of Medicine. 156 pp. illust. $5.75. Charles C. Thomas, Springfield, Ill., The Ryerson Press, Toronto, 1953.

This is a monograph which accomplishes in an admirable manner what it was designed to do. It covers the development of obstetrical forceps and their use from the time of their invention to the present day. It is an attractive little book, well bound, printed in clear type on good quality paper, the format is pleasing, and above all, it is exceptionally well written. The author is obviously a master of his subject, and an excellent teacher.

The statement that the blade of the Kjelland forceps, like the Barton, possesses no pelvic curve, is hardly correct. If the author will superimpose the blade of a Barton forceps on that of a Kjelland, he cannot fail to note the presence of the curve in the Kjelland blade.

In chapter V, on the Mechanism of the Forceps, the author mentions the "toe hold" which the tip of the forceps obtains when it anchors under a bony prominence". It might be better to call this the chin grip, because the *chin* is the bony prominence under which the blades *should* anchor, not the maxilla or the mandible. One cannot agree with the statement that "anchorage under either mandible or maxilla is satisfactory". Traction with the blades grasping the maxilla particularly, transmits a compression force to the floor of the cranial vault, and may cause fracture of the floor and possible trauma to the respiratory centre. The application in Fig. 22 is not good, the one in Fig. 23 is bad. The tip of the blades should go to the chin.

Chapter VI, Application of the Forceps. The caption for Fig. 35 states (2) The posterior fontanelle is one fingerbreadth above the plane (of the shanks); This is as it should be, but the illustration unfortunately shows it below. Fig. 37 shows the relationship of the shanks and upper margins of the blades to the lambdoidal sutures to be much too high. The application of blades shown in Fig. 38 is bad. The tip of the blades should be at the chin.

Reference is made to the universal joint in the Tarnier instrument. There is no universal joint in this forceps. The traction rods are attached to the blades by a simple hinge joint. The other swivel joints do not combine with this to produce the mechanical contrivance known as a universal joint. The author's defence of the Tarnier Forceps as an "automatic pilot for the pelvic channel", in spite of his itemized objections to the instrument, is difficult to justify.

Mr. Malloy's illustrations throughout are first class. If he hadn't used so much black on the operators' hands they would be much more attractive. Apart from the criticisms mentioned, this is an excellent book and is to be highly recommended, to the student beginner, the interne, the practitioner and to the highly skilled specialist.

THE PHOTOGRAPHY OF PATIENTS

H. L. Gibson, Medical Division, Eastman Kodak Company, Rochester, New York. 118 pp. illust. $3.50. Charles C. Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1952.

"The Photography of Patients" is a neat little volume, number 95 in the American Lecture Series. It is well-presented and illustrated but unfortunately skims blithely over only a few phases of the subject and this with a decided Eastman Kodak perspective and promotional flavour. Photography is photography, whether of patients or other people. It is a profound subject, confusing and detailed whether as a hobby or as an aid to one's professional endeavours. In other words, there is no short-cut to a working knowledge of photography and this volume may have a few points for the novice and amateur but has nothing for the advanced photographer.

AN ATLAS OF SKULL ROENTGENOGRAMS

B. S. Epstein, Associate Radiologist, The Jewish Hospital of Brooklyn, Brooklyn, New York; and L. M. Davidoff, Neurosurgeon, to the Mount Sinai Hospital, New York City. 415 pp. illust. $16.50. Lea & Febiger, Philadelphia; The Macmillan Company of Canada, Toronto, 1953.

This book is truly an atlas. Less than 60 of its 410 pages do not contain reproductions of skull roentgenograms or photographs of diseased structures. There are over 600 illustrations, usually two to a page, all of which are as large as practical for the size of the book. The technical and photographic difficulties involved in projecting details of two sides of an oval osseous structure such as the cranial vault on to a plane surface is well known to all those interested in Radiology. The collection of films in this book must have been carefully selected as to quality in order to obtain the clear printed reproductions. While radiographic detail must necessarily be sacrificed in the process of double printing, radiographic contrast has been preserved to a remarkable uniformity throughout the book.

The illustration captions are short but explanatory, and more than offset the brevity of the text which serves more as an introduction to the varied collection of films featured in each chapter.

The book is essentially a study of the plain film of the vault and base of the skull. It demonstrates x-ray patterns of the normal skull, developmental anomalies, traumatic and infective processes, neoplasms of the brain and skull, as well as a large collection of films showing other diseases of the skull. Planigrams, radiographic and photographic illustrations of pathological specimens are used freely to supplement the abnormality shown in con-

ventional roentgenograms. Cerebral pneumography is featured only occasionally to accentuate findings in the plain film.

Diseases of the accessory nasal sinuses and mastoids are not included. There is no bibliography, but the greater portion of the book is made up of the authors' original material.

This atlas, together with the authors' preceding text, "The Abnormal Encephalogram", make an excellent combination for the radiologist's reference library. The atlas will be most useful for students and physicians who wish to study a systematic demonstration of diseases of the vault of the skull.

HOLT PEDIATRICS

L. E. Holt, Jr., Professor of Pædiatrics, New York University College of Medicine; and R. McIntosh, Carpentier Professor of Pædiatrics, Columbia University, and Director of the Pædiatric Service in the Babies Hospital, New York City. 1485 pp. illust., 12th ed. $15.00. Appleton-Century-Crofts, Inc., New York, 1953.

With the development of Pædiatrics by groups of investigators interested in various aspects of the specialty, this book has taken on multiple authorship under the guidance of Dr. Holt and Dr. McIntosh. Their collaborators have been drawn from medical schools in widely separated parts of the country and include a distinguished group of authorities, well equipped to present the special fields to which, in many cases, they themselves have made notable contributions. The work of these contributors is well drawn together and differences in points of view are not apparent.

The arrangement of the book has been thoughtfully done and the sequence of the presentation is good. Chapters are divided and sub-divided by heavy type titles, a system which will appeal to the medical student and busy practitioner. For those who need to go beyond the text an excellent list of references is provided at the end of each section. These are carefully chosen and of recent date, many of them as late as 1953. At first glance the book appears to be too brief, but as one uses it one becomes impressed by its completeness and how infrequently it fails to provide either in itself, or in its bibliography, the material for which one has consulted it. One can ask no more of a good textbook. Certainly the book has maintained the high quality set by the original authors and may be recommended to medical student, general practitioner and pædiatrician alike.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Physical Medicine and Rehabilitation. Edited by B. Kiernander, Director, Physical Medicine Department, Hospital for Sick Children, Great Ormond Street, London. 610 pp., illust., $15.00. Charles C. Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1953.

Reason and Unreason in Psychological Medicine. E. B. Strauss, Physician for Psychological Medicine, St. Bartholomew's Hospital; Lecturer in Psychological Medicine, St. Bartholomew's Hospital Medical College (University of London). 55 pp., $2.75. Charles C. Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1953.

Sience and Man's Behavior. T. Burrow; Edited by W. E. Galt. 564 pp., illust., $6.00. Philosophical Library, New York, 1953.

Les Cahiers de l'Hôtel-Dieu de Québec. Chroniques Médico Hospitalières. 554 pp., illust., Septième Année. Charrier & Dugal, Inc., Québec, 1952.

A Source-Book of Medical Terms. E. C. Jaeger, Formerly Head, Department of Zoology, Riverside College, Riverside, California. 145 pp., illust., $6.00. Charles C. Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1953.

A Handbook on Diseases of Children. B. Williamson, Physician, Children's Department, Royal Northern Hospital, London; Physician, Children's Hospital, Northaw. 467 pp., illust., 7th ed., $3.60. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Co. of Canada Ltd., Toronto 2, 1953.

The Psychiatrist, His Training and Development. Report of the 1952 Conference on Psychiatric Education held at Cornell University, Ithaca, N.Y., June 19 to 25, 1952. Edited by J. C. Whitehorn, and others. 214 pp., illust. American Psychiatric Association, Washington, 1953.

The General Practitioner's Guide to Physiotherapy. Janet Dennison, Hon. Adviser, Physiotherapy Department, East Surrey Hospital, Redhill. 38 pp., $1.25. William Heinemann Medical Books Ltd.; British Book Service (Canada) Ltd., Toronto 6, 1953.

Medical Electronics. G. E. Donovan. 215 pp., illust., $6.50. Butterworth & Co. (Canada) Ltd., Toronto 6, 1953.

Applied Cytology. G. R. Osborn, Pathologist to the Derbyshire Royal Infirmary and Derbyshire Hospital for Women; Honorary Lecturer, Department of Pathology, The University of Sheffield. 168 pp., illust., $7.00. Butterworth & Co. (Canada) Ltd., Toronto 6, 1953.

Expert Committee on Nutrition. World Health Organization: Technical Report Series No. 72. 30 pp., 3rd report, $0.20, Fr. fr. 65.-, Sw. fr. 0.80. Also available in French ed. World Health Organization, Palais des Nations, Geneva; The Ryerson Press, Toronto, December 1953.

Influenza: A Review of Current Research. World Health Organization: Monograph Series No. 20. 224 pp., $2.50, Sw. fr. 10.--, 17/6d. French edition in preparation. World Health Organization, Palais des Nations, Geneva; The Ryerson Press, Toronto, 1954.

Advances in the Control of Zoonoses. World Health Organization: Monograph Series No. 19. 276 pp., $3.00, Fr. fr. 960.-, Sw. fr. 12.-, 15/-. French edition in preparation. Also published as FAO Agricultural Studies No. 25. World Health Organization, Palais des Nations, Geneva; The Ryerson Press, Toronto, 1953.

Clinical Genetics. Edited by A. Sorsby, Research Professor in Ophthalmology, Royal College of Surgeons of England and Royal Eye Hospital, London. 580 pp., illust. $18.00. Butterworth & Co. (Canada) Ltd., Toronto 6, 1953.

The Faber Medical Dictionary. Edited by Sir C. Wakeley, Fellow of King's College, London, President of the Royal College of Surgeons of England. 471 pp., $9.00. Faber and Faber Limited, London; British Book Service (Canada) Ltd., Toronto 6, 1953.

The British Contribution to Medicine. J. Jaramillo-Arango, Former Rector of the National Faculty of Medicine of Bagota, Former Colombian Ambassador in London. 204 pp. Illust. $4.25. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Ltd., Toronto 2, 1953.

A Textbook for Midwives. M. F. Myles, Until Recently Principal Midwife Tutor, Simpson Memorial Maternity Pavilion, Edinburgh. Formerly Director of Education, Woman's Hospital, Detroit, U.S.A. 676 pp., Illust. $7.15. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Ltd., Toronto 2, 1953.

Peptic Ulcer. C. F. W. Illingworth, Regius Professor of Surgery, University of Glasgow, Surgeon, Western Infirmary, Glasgow. 287 pp. Illust. $7.15. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Ltd., Toronto 2, 1953.

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References: in the case of a journal arrange as follows: author (JONES, A. B.), title, journal, volume, page, year. In the case of a book: WILSON, A., Practice of Medicine, Macmillan, London, 1st ed., p. 120, 1922.

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If a box number is requested, there will be an additional charge of 50c on the first advertisement to cover postage and handling charges.

NOTICE.—EDINBURGH POST-GRADUATE BOARD FOR MEDICINE. General surgery. Three months' courses of post-graduate surgery are arranged to start on March 22nd and September 27th, 1954. These are suitable for surgeons requiring refresher courses in the current outlook on general surgery, or for graduates preparing to specialise in surgery; approximately 275 hours of instruction are provided. Fee £31:10/-. Medical sciences. A three months' course in applied anatomy, physiology, pathology, bacteriology and biochemistry will begin on June 28th, 1954. This course is suitable for post-graduates wishing to take the Primary Fellowship Examination, as a final preparation in these subjects. Considerable basic knowledge is highly desirable prior to taking this course. Fee £31:10/-. A similar course of 10 weeks' duration is organised by the Royal College of Surgeons under the ægis of the Post-Graduate Board, starting on the 4th of October, 1954. Fee £26:5/-. Internal medicine. Courses lasting 12 weeks, suitable for graduates wishing a refresher course, or to specialise in medicine, begin on March 29th and September 27th, 1954. These courses consist of 320 hours instruction, comprising lectures, clinical demonstrations and ward visits. Fee £31:10/-. Applications for enrolment should be addressed to the Director of Post-Graduate Studies, Surgeons' Hall, Edinburgh 8, supplying particulars of qualifications and post-graduate experience.

FOR RENT.—Medical office at 948 Main Street East, Hamilton, Ontario. Has common waiting room with dermatologist, excellent location for another specialist. Apply Dr. L. T. Williamson, at above address.

FOR RENT.—Spacious office suitable for certified pædiatrician, in Guelph, Ontario, a city now without a pædiatrician, and with excellent hospital facilities. Apply to Box 788, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

FOR SALE OR RENT.—Long established general practice at Cookstown, Ontario. Combined residence and office, modern conveniences. Paved highways, close to open hospital at Barrie and Alliston. Splendid agricultural community. Office equipment and supplies available, if desired. Apply Dr. Chas. Carr, Cookstown, Ontario.

FOR SALE.—Doctor's combined office and house for sale due to recent bereavement, in Kemptville, Ontario. Brick house, oil heating, and office equipped. One licensed private hospital and one nursing home; industries; agricultural school; Bordens Limited; Moore Business Forms; Dept. of Lands and Forests; Dominion Concrete. Situated on Ottawa Prescott Highway. Available immediately. Can be purchased on terms. Apply to Mrs. G. D. Gordon, Kemptville, Ontario.

FOR SALE.—Prescription bottles, Imported materials from England at import prices direct to you. All sizes. Write for prices to Import Specialties Limited, Bracebridge, Ontario.

FOR SALE.—General practice in growing Alberta town drawing from farming district and nearby lumbering industry. A 25-bed modern hospital serves this community. 6-room well-equipped office. Annual income $25,000. Owner, who wishes to specialize, will help buyer establish. Terms can be arranged. Apply to Box 774, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

FOR SALE.—Unopposed general practice in central Saskatchewan town with modern 16-bed hospital. Fully equipped office with laboratory, diathermy, B.M.R., etc. Modern 6-room residence with all conveniences. Gross income between $23,000 and $24,000. Fair value $13,000. Available June 1954. Apply to Box 785, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

FOR SALE.—Established medical office for 21 years, located in the most busy street in a flourishing city in Ontario. Apply to Box 790, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

FOR SALE.—Opportunity for gracious living in a rural and urban general practice. Good fishing, hunting, skiing, golfing, swimming, sailing etc. Good schools and shopping. House and office combined. All modern conveniences and major maintenance, up-to-date. Two open hospitals. Opportunity to do own and referral surgery. Practice established 70 years. 100 miles from Toronto. Owner specializing in July 1954. Apply to Box 792, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

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Thirty Years Ago . . .

From the *Journal* of March, 1924

FROM AN ADDRESS BY DR. W. W. CHIPMAN, "THE OPENING OF THE WOMAN'S CLINIC JOHNS HOPKINS HOSPITAL."

"One of the first advisers, the wise and energetic John S. Billings, in his address at the opening of this hospital, remarked that, 'hospitals are in some sort the measure of the civilization of a people, or even of a city.' In this way Johns Hopkins affords but another proof that the citizens of Baltimore are indeed equal to the best; and that this fair city may justly be proud of its own civilization. At the time of his retirement, and when the hospital was but thirteen years of age, President Gilman wrote lovingly of it as, 'doubling the Cape of Good Hope, which leads to a Pacific Sea in whose bounds are the Fortunate Isles.' Doubling the Cape two-and-twenty years ago! During these intervening years, his beloved hospital has voyaged far within that Pacific Sea, and has reached today one of the very desirable of those Fortunate Islands."

NOVA SCOTIA NEWS

"A report on the vital statistics of Nova Scotia for the month of September, 1923, recently issued by the Department of Public Health, indicates that the general death rate for the month was 10.3; the infant mortality rate was 80.2; and the tuberculosis (all forms) rate was 103."

NEW BRUNSWICK NEWS

"At a meeting of the profession held recently in Saint John, the proposal of the Government to do away with medical superintendents in the D.S.C.R. hospitals was discussed. Insufficient information was available to thoroughly understand the proposed changes, but it was unanimously decided to protest against a lay man replacing a physician as Superintendent. Telegrams to that effect were sent to several of the Ministers at Ottawa."

BRITISH COLUMBIA NEWS

"The deep interest shown by members throughout the province in the activities of the British Columbia Medical Association, speaks well for its success during 1924 and is encouraging to the Executive which has had many problems, chiefly of an economic nature, to deal with. A recent trip of the Executive Secretary to Vancouver Island, when a personal visit was made to practically every doctor in Victoria, Duncan, Ladysmith and Nanaimo proved most gratifying in results. Membership fees were cheerfully paid and the keenest interest taken in the Association's activities.

"A special bulletin is in course of preparation and will shortly be in the hands of each member.

"The third monthly luncheon of the British Columbia Medical Association was held on January 22, when an attendance of 110 medical men listened to an able address from Mr. E. S. H. Winn, Chairman of the Workmen's Compensation Board, on 'Health Insurance'.

"Several Victoria doctors have expressed themselves as particularly interested in the Health Insurance Scheme and have offered their whole hearted support and assistance to the central committee.

"The next monthly luncheon of the British Columbia Medical Association will be held, probably early in March, when it is hoped that the Hon. Dr. J. D. MacLean, Provincial Minister of Education, will give an address on matters peculiarly affecting the profession."

NEWS AND NOTES

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1954 BRITISH INDUSTRIES FAIR

The thirty-third annual British Industries Fair will be held in London (Earls Court and Olympia) and Birmingham (Castle Bromwich), from May 3 to May 14, 1954.

The B.I.F. is one of the more dramatic means by which the U.K. Government is seeking to expand its export trade—a vital task, for on its success depends Britain's ability to pay for her imports, including her substantial purchases from Canada.

At the Fair, the overseas buyer has only to show his official invitation or business card to receive a badge admitting him freely at any time to all three sections, and to the Overseas Buyers' Clubs. Visitors are encouraged to use the clubs as their headquarters, where mail can be addressed, and to make use of secretarial, telephone, cable and radio facilities.

FEDERAL GRANT TO MANITOBA TO TRAIN LAB TECHNICIANS

Bursaries are being provided with assistance from the federal government for technicians training at Brandon Hospital to staff Manitoba's diagnostic services. Approval had been given to a grant to the province of $8,772.85 for this purpose from the Laboratory and Radiological Service Grant under the National Health Program.

Manitoba is in urgent need of technicians and is training addiitonal staff as speedily as suitable personnel can be recruited. Five additional students entered the course in August with assistance from the federal grant.

NEW FEDERAL HEALTH GRANTS AID NOVA SCOTIA SERVICES

Additional grants totalling more than $10,000 have been made under the National Health Program in support of professional training for the health services of Nova Scotia. These funds will make possible the employment of additional professional staff and extension of the provinces health program in several fields.

Ottawa will pay the province $3,711.51 for training three physio and occupational therapists, who are taking two year courses in their field, two at University of Toronto and one at McGill University. It was indicated that there will probably be many such positions to be filled in Nova Scotia within the next few years, particularly in view of the possibility of extension of the province's rehabilitation program.

Two dental hygienists are to receive special training, one at the Eastman Dental Dispensary, Rochester, N.Y., and on at the University of Toronto, in a move to provide ancillary personnel to implement the province's dental health activity, which has been speeded up due to increased interest in the field through health education of children and the institution of a topical fluoride preventive program. The federal government will contribute $2,688.50 for this project.

The National Health Program's professional training grant will provide $2,250 as a bursary for an Ontario physician who is taking postgraduate training at the School of Hygiene, University of Toronto, before assuming an appointment in Nova Scotia.

Also, under the Program's Child and Maternal Health grant, Ottawa will pay $720 to cover expenses of 16 nurses to attend a course designed to deal with basic modern concepts and techniques with regard to prenatal care.

A fifth approved grants project provides Nova Scotia with $700 to cover the cost of a short course in bronchoscopy for a provincial medical officer at the Philadelphia Bronchoscopy Clinic. As a member of the Nova Scotia Sanatorium's travelling surgical and consulting team, the doctor will later be enabled to carry out certain bronchoscopic examinations in the field.

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NEWS AND NOTES

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SOME REFLECTIONS UPON THEORY AND PRACTICE*

By Frank Hebb, M.D.

No thinking individual lacks due respect for the advances of engineering and the laboratory. However, in this age of fascination with mechanics should anyone step over the line into, say, the field of abstract thought or roam across the boundaries of compartmentalized learning, he may then be looked upon as eccentric. Let him go beyond the realm of specialization and fail to adhere to irreducible and stubborn facts and he is classified as impractical. The practical man in other words, seems to have about all the genius which this age can afford. The long and short of it is that modern man has no need of a ghostly metaphysical world inhabited by beautiful theories. Like the musical banks in Samuel Butler's *Erewhon*, they make a delightful noise but they cash no cheques.

No modern doctor, however, wishes to return to the abstruse philosophical climate of the Middle Ages. They had few or no statistics, which was an unquestionable handicap. But we, their successors, are also handicapped the moment we discount the value of any convictions not directly correlated with factual data. . . Take for example one of our most frequent pitfalls, that of "type-casting". It is a reliance on "the familiar" and can be found wherever there is an obvious technical advantage in classifying human beings. The extensive personnel testing of persons who apply for work in large-scale industry illustrates the growing habit of "typing" people according to their superficial attributes. There is no real harm of course in trying to determine people's special capacities but inevitably the factors which are important to an employer of labour assume greater importance than the human qualities of individuals. The tests are devised to anticipate the probable dollars-and-cents value to a prospective employer of a man or woman at a given occupation. Often, however, larger implications than the "skill" or "temperamental traits" of the tested individuals are read into the results of these tests. The psychology of the tests also, is largely in terms of fixed norms or values. They seek to establish what a man *is*, not what he may be trying to *become*. Their general effect on human beings therefore is in terms of "status-quo-ism".

This attitude is not only flourishing in many places but seems to be doing so with the full consent of the psychologist. True, intelligence tests have been improved in recent years; but they will become significant only when the results of the tests are given in their proper context. The affirmation that A's intelligence quotient is higher than B's tells us, as it stands, very little. But one must admit, that, imperfect as it is, it has done something to give statistical form and content to the universally held conviction that some people are stupider than others. Today, a lazy student who receives a failing grade is likely to be diagnosed as maladjusted. Similarly, the "well-adjusted" personality rates high in any listing of virtues. Then the term "well-integrated" personality is beginning to appear on recommendations. It could be that well-adjusted people are those who never give any trouble, well-integrated may mean only a person without any individuality or ideas. . . . A story is told of a querulous psychologist who underwent 221 hours of psychoanalysis for a Rockefeller Foundation inquiry. During it the psychologist asked his analyst "What is normality?" "I don't know," the analyst replied. "I never deal with normal people." The psychologist persisted: "But suppose a really normal person came to you?" The analyst admitted: "Even though he were normal at the beginning of the analysis, the analytic procedure would create a neurosis."

*Reprinted from *The Bulletin of the Vancouver Medical Association*, November, 1953.

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NEWS AND NOTES

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. . . In short, Medicine can never become an exact science unless all the variables can be estimated and allowed for, unless the personal element can be eliminated and unless each patient can be reduced to a standard form. As things are, the normal variations in individuals have such a wide range that both the automatic interpretation of facts and the mechanical prescription of treatment are prohibited. John Ryle wrote "variability is one of the most distinctive and necessary attributes of life which admits of no constant and no norm."

Only too often as medical practitioners our vision becomes so restricted that we can see only a single means of curing ailments, a certain form of diet, a particular medicine, some electrical method of treatment, or an ingenious operation. Health entails, after all, a balancing of all our functions as well as adjustments between ourselves and the outside world. If this balance and adjustment is upset, it can rarely be restored by the use of any single remedy. . . . When Max Planck was invited to describe the role of Science in human life he replied that Science was "a constructed work of art expressing a certain side of Man's Nature". This definition acknowledged that there are many sides to Man's Nature which cannot find expression in Science. We have not only a scientist within us, but also a poet, an artist and even traces of a potential saint. It is for each of us to discover how the growth of the less developed sides of our nature can be encouraged so that we may live life richly and well. Eric Gill once said that an artist is not a special kind of man, but that every man is a special kind of artist. Indeed, too many of us think of everything in terms of our own science and turn a blind eye to everything else. There is a rather pathetic entry in Charles Darwin's diary—"My mind seems to have become a kind of machine for grinding general laws out of large collections of facts but why this should have caused the atrophy of that part of the brain alone, on which the highest tastes depend, I cannot conceive. . . . The loss of these tastes is a loss of happiness, and may possibly be injurious to the intellect and more probably to the moral character by enfeebling the emotional part of our nature."

One recalls medical school days, during which time we were required to burden our memories with so large a number of relevant facts that we simply dare not take the risk of taxing our minds with too much extra curricular reading or thinking, for fear lest it crowd out some of the knowledge of importance. Pre-eminently this was a period in one's life, eighteen to twenty-three years of age, when future tastes and interests are formed. . . . Today it is heartening to find a decided step forward in some medical schools where opportunity is given for the development of a more liberal education. Deprived of this as a youth, a doctor remains limited as a man on account of the arduous conditions of hospital and private practice which preclude the chance of repairing the limitations which lack of an all-round education has imposed. In the August, 1952 issue of *The Canadian Doctor* the editor has this to say: "The major concern of the doctor of tomorrow will be to look upon a patient as a 'whole human being'. Unless we limit ourselves to research in a special field we must be careful to avoid the development of compartmentalized minds. It has never been enough for any doctor to confine himself to the context of textbooks of medicine. At the present time we are more aware than ever that artificial barriers to thought must be broken down, not only within the science of medicine itself, but also where they tend to isolate medicine from related Arts and Sciences. The ultimate goal after all is, as Myerson stated it, 'to understand Man'."

Science can satisfy many of our needs but not those of the Spirit. If our sense of the meaning of things is not developing then we are not developing; for what is left of a man when you leave out his ideas and feelings about the meaning of life? In medicine we might do well to study the fact, the theory, the alternative and the ideal, *i.e.*, stretch the mind in all directions in order to avoid falling into unsuspected provincialisms of opinion. The modern sage A. N. Whitehead has written that philosophy asks the simple question "What is it all about?" He defines speculative philosophy as the endeavour to construct a system of general ideas "in terms of which every element in our experience can be interpreted." This does not mean that there should be less study of work and more study of thought, *i.e.*, educational escapism, where education becomes an end in itself: . . . rather, that we make a new and vigorous effort to foster in ourselves a wise philosophy of the science and the art of medicine, in the hope that there may result therefrom the greatest of all gifts that the physician can possess—understanding.

Lastly, this article is far too short for so vast and complex a subject and it goes without saying that the task has been inadequately performed. However, I make no excuses for attempting it. A brief theory is better than no theory at all.

APPROVE FEDERAL GRANT FOR ALBERTA HOSPITAL ADDITION

A grant of $21,750 towards construction of an addition to St. Joseph's Hospital, Galahad, Alta., has been approved under the Hospital Construction Grant in the National Health Program.

The grant contributed to the cost of providing 40 patient beds at the hospital operated by the Sisters of St. Joseph at Galahad and serving a population of 5,500. With completion of the addition, a section of the hospital formerly used for patients will accommodate members of the nursing and administrative staffs as well as a chapel. This old section formerly accommodated 15 treatment beds, so the net increase in the hospital will be 25 active treatment beds. The federal health grant is based on $750 per active treatment bed and a like amount for one labour bed as well as $2,250 for nine bassinets in cubicles.

The establishment of a rehabilitation camp for chronic alcoholics by the City of Vancouver was urged recently at a hearing before the Vancouver Police Commission. City field worker George Slater said that a trial camp last spring succeeded in rehabilitating 3 of 21 chronic drinkers and declared that a camp with adequate supervision would bring greater gains on the problem. Judge W. H. S. Dickson of the Family Court has urged that Mr. Slater's services be made available to the court as counsellor on family alcoholic problems. . . . Plans set up by the Alcoholism Foundation of Alberta will give

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NEWS AND NOTES

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Calgary a branch of the Foundation early in 1954. G. George Strachan of Edmonton, executive director of the Foundation, has stated that centres will also be opened in all major cities in the province to deal with the public health problem of alcoholism. The Foundation will work in close co-operation with Alcoholics Anonymous and provide skilled medical and clinical help. One problem faced by the Foundation was in obtaining trained personnel for the work. He stated that Calgary has a minimum of 1,500 alcoholics.

PSYCHIATRIC SOCIAL WORK INSTITUTE HELD

Said to be the first of its kind in Canada, a medical and psychiatric social work institute was held recently in Montreal at the Jewish General Hospital, the Montreal General Hospital and the Royal Victoria Hospital on succeeding days under the auspices of the Eastern Canada district, American Association of Medical Social Workers. The general theme of the Institute was "Teamwork in the Medical Setting" and registration anticipated at 75, rose to close to 180 and included 30 U.S. workers. Representatives came from Ontario, Quebec and the Maritimes and the registration had to be closed three weeks before the Institute opened. Many late applications had to be refused owing to lack of space and also to the fact that the discussion method used throughout could not easily be utilized for large groups.

From the letters of commendation which have come in since the Institute was held, reports Miss Avis Pumphrey, director of the Social Service Department of the Montreal General Hospital, "we feel that it must have met a real need."

The first day's session was held in the nurses' residence of the Jewish General Hospital and introductory talks were given by Miss Pumphrey, chairman of the Eastern Canada District and Miss Alice Lepine, the latter speaking in French. The morning session was spent discussing teamwork, both effective and ineffective, resulting from (a) good and bad recording and reporting and (b) good and bad interpretation to the medical team. Basis for the discussion was a socio-drama presented by local service staffs. In the afternoon, the meeting dealt with teamwork in rehabilitation within the hospital as exemplified in the program for cerebral palsy patients at the Children's Memorial Hospital. Mrs. Elizabeth Dunlop was chairman and the panel included Dr. Preston Robb, senior neurologist and medical director of the Cerebral Palsy Clinic, psychiatrists Dr. Taylor Statten and Dr. Hyman Caplan, and medical social worker Miss Barbara Allen.

CANADIAN SOCIETY FOR THE STUDY OF FERTILITY

Research and therapy in human reproduction are fundamental needs today. Reproductive wastage probably outranks all other causes of death—one in ten couples are involuntarily infertile. Each year in the U.S.A. alone over 300,000 desired children are not born and not even conceived, one in five pregnancies end in spontaneous abortion or miscarriage— over 400,000 spontaneous abortions and miscarriages occur yearly in the U.S.A. alone. Death due to premature birth occurred over 31,000 times in the U.S.A. in 1945. ("See Research in Human Reproduction", a pamphlet prepared by the Planned Parenthood Federation of America Inc.—in collaboration with the National Committee on Maternal Health and the Committee on Human Reproduction of the National Research Council.)

Nine physicians and two doctors of veterinary medicine met in Toronto on May 5, 1953, to form a Committee for the Study of Sterility. A further organization meeting was held in New York City on May 27 during the First World Congress on Fertility and Sterility. This meeting was attended by 26 physicians and two doctors of veterinary medicine from seven provinces.

The aims of the Society are to promote investigative and educational work in the field of reproduction; to evaluate diagnostic methods and therapy.

The Canadian Society for the Study of Fertility was founded officially at a meeting of the Board of Directors in Toronto July 1, 1953. The following are the officers—President, L. J. Harris, M.D., F.R.C.S.[C], Toronto; Vice-President, M. M. Braunstein, M.D., Montreal; Secretary-Treasurer, R. J. McDonald, D.V.M., Woodstock, Ont.; Directors, E. V. Shute, M.B., F.R.C.S.[C], London, Ont.; Prof. C. A. V. Barker, D.V.M., M.Sc., Guelph, Ont.; Prof. J. Ross Vant, M.D., F.R.C.S.[C], Edmonton, Alta.; Joseph Tanzman, M.D., St. John, N.B.

The First Annual Meeting of the C.S.S.F. will be held in Montreal on October 1, 1954.

Inquiries should be addressed to the secretary.

AMERICAN BOARD OF OBSTETRICS AND GYNÆCOLOGY

The next scheduled examinations, Part II (oral and pathological), for all candidates will be held at the Edgewater Beach Hotel, Chicago, Illinois, May 10 to 17, 1954. Formal notice of the exact time of each candidate's examination will be sent him several weeks in advance of the examination.